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1861. Whitelaw, Wm., M.D., Kirkintilloch, Dumbartonshire.

Jenner Medallists.

1898. William Henry Power, C.B., F.R.S., Chief Medical Officer of the Local Government Board, Whitehall, S.W.
1902. A. Laveran, Membre de l'Institut et de l'Academie de Medicine, 25, Rue du Montparnasse.

TRANSACTIONS

OF

The Epidemiological Society of London.

WHAT IS CLIMATIC DISEASE?

By LIEUTENANT-COLONEL A. M. DAVIES, R.A.M.C.

(*Read: November* 21*st*, 1902.)

THE few remarks that follow are intended merely to introduce the question, What is Climatic Disease?

In former times people used to speak of the deadly climate of Calcutta, or Jamaica, or other tropical places; and, until about the middle of the last century, it must be admitted that this was true in the case of many of our foreign stations. Thus, the death-rate of the British soldier in India averaged (up to 1855) 69 per 1,000, the rate at home being 9 per 1,000 among men at the soldier's age. But when the causes of this high mortality were enquired into, it was found that by far the largest number of deaths were due to a few diseases of the zymotic class; so that, when the Indian Sanitary Commission presented their report in 1863, they were able to say that, "were it possible wholly to put an end to deaths arising from the various classes of fevers, from cholera, dysentery, amd diarrhœa, together with hepatitis and its consequences, the troops serving in India would become as healthy as any body of men in the world." The Commissioners pronounced the opinion that these diseases were, to a large extent, "dependent on removable conditions and habits." I may mention that one of these habits was the drinking of the fourth part of a quart bottle of raw spirits every day, as a ration; and another, the marching in a close-fitting tunic and stock, tightly buckled across the chest, and the head very imperfectly protected from the sun.

It was from this time probably that may be dated any general or widespread correctness of view with regard to tropical hygiene and disease causation; for Dr. Parkes, writing a few years later, still thought it might seem "a bold thing to question the commonly-received opinion, that

a tropical climate is injurious to a northern constitution." The specific character of the zymotic diseases was well known, though the actual nature of the specific *contagia* was, of course, not then recognised; but while asserting that climate was not the real *causa causans* in this class of diseases, Dr. Parkes was still able to hold that "when a climate is called 'unhealthy,' in many cases it is simply meant that it is malarious." And no doubt most authorities would have agreed with this until the other day, that is, a few years ago; for, although Laveran published his observations on the plasmodium in 1880, but little attention was paid to them for some years (Dr. Bristowe, for instance, did not allude to them in his last edition, 1890); and only since about 1896, when Dr. Manson changed the whole practical aspect of malaria causation and prevention, has medical opinion generally recognised that these fevers, as well as the zymotic group, are not *essentially* dependent on any of the factors that go to make up climate.

What affections then, if any, are left to be included, strictly speaking, under the heading of Climatic Diseases? Besides "hepatitis and its consequences" (as to which, however, many authorities would be doubtful), apparently there would be nothing but sunstroke and frostbite. But three years ago Dr. Sambon wrote a most interesting paper on the first-named of these, and brought much evidence to show that it was due to a specific poison. This view is said by Osler to be "one worthy of most careful study," and by Manson to have "more in its favour than any of the purely thermic theories." If accepted, the group of climatic diseases becomes reduced almost to vanishing point.

It must be admitted that those diseases bearing local names, such as Malta fever, Roman fever, Peshawar fever, and so on, are not essentially climatic, being either local manifestations of malarial fever (Peshawar fever, etc.), or typhoid fever *plus* malaria (Roman fever), or the separate and distinct specific disease, Mediterranean fever, or the "non-malarial remittent" of Crombie (Calcutta fever, etc.), the specific cause of which has not yet been differentiated, though it may turn out to be *Bacillus coli.*

There are, however, some other diseases that must, I think, still be held to be essentially climatic. Of what Hirsch calls "the trio of tropical diseases in the stricter sense of the term," malarial fever, suppurative inflammation of the liver, and dysentery, the two latter have not up to the present been definitely shown to be due to a specific

poison, while their dependence on climatic conditions is undoubted.

Tropical liver abscess appears to be due especially to extreme *fluctuations* of temperature, causing *chill, i.e.*, cold nights following on hot days, such as are experienced in the rainy season in India, and especially in the more elevated stations in Madras Presidency (Secunderabad, Bangalore) and Upper Burmah (Shwebo, Thayetmyo): the beginning of the night is hot, and the tendency is to throw off all clothing; in the early hours of the morning there is a sudden drop in temperature, and the abdomen becomes chilled before one becomes sufficiently aware of the cold to cover oneself up. This would seem to be a strictly climatic determining cause. The same extreme fluctuations in temperature occur in Senegal, where the day temperature may be 104 deg., while in the night it may fall to 60 deg. Liver abscess and hepatitis are rare in the West Indies, where the climate, though tropical, is equable.

Of dysentery, also, Hirsch says, "that though a somewhat high temperature is an essential factor in the etiology, the worst time in an endemic prevalence is when there is extreme fluctuation between hot days and cold nights." There is, of course, a close relationship between dysentery and liver inflammation and abscess, also with malaria. Without entering on this topic, everyone will admit that *chill* is a most powerful factor in bringing about an attack in each disease; but while in the case of liver abscess this chill appears to be always a *tropical* chill, in the case of dysentery it cannot be said that a tropical climate is necessary for its endemic prevalence; since in Sweden, Iceland, and Denmark it has been endemic; also in Ireland formerly; while epidemics of asylum dysentery (if this be the same disease) have occurred in this oountry quite lately. There is no necessity for a tropical climate in regard to this disease. As to the true relations of the so-called *Amœba dysenteriæ*, opinions are divided; it appears certainly not to be the invariable cause, therefore not the essential cause. The same may probably be said with regard to the other organisms—bacteria belonging to the *coli* group—that have been found to be present in various epidemics.

Now, with regard to specific diseases, Dr. Manson has put the question very clearly: "In the tropics, as in temperate climates, in the European and in the native alike, nearly all disease is of specific origin. It is in their specific causes that the difference between the diseases of temperate climates and those of tropical climates princi-

pally lies." The germs of disease demand certain physical conditions for their well-being; one of these is a certain temperature. If the temperature of the transmitting medium is too high or too low, the germ ceases to be infective: so may be explained the absence from the tropics of a class of directly infectious diseases, represented by scarlet fever, and the absence from temperate climates of a similar class of diseases, represented by dengue.

Again, many diseases require for their transmission the services of a third and different animal. If this be a tropical species the disease must be confined to the tropics, *e.g.*, tsetse-fly disease and filariasis.

Certain parasites pass a part of their lives as free organisms in the outer world, where they require a relatively high temperature for their development; they must, therefore, be tropical or sub-tropical, *e.g.*, *ankylostomum duodenale* and *ankylostomiasis.*

Again, disease organisms may have their natural enemies, and the geographical range of such disease-germs will depend not only on the presence of favourable, but on the absence of unfavourable, conditions.

So that "the more we learn about these diseases the less important in its bearing on their geographical distribution, and as a direct pathogenic agency becomes the *rôle* of temperature *per se*, and the more the influence of the tropical fauna."

Of diseases which, though dependent on a specific cause, are in addition essentially linked with a tropical climate, yellow fever is a marked example; as a permanent disease, lasting, that is to say, the whole year through, it is found only where the mean winter temperature is at least 68 deg. to 72 deg. Fahr.; it has never grown into an epidemic except when the heat has equalled the mean annual temperature of the tropics (75 deg. Fahr.), and it has on no occasion become diffused in a temperature below 68 deg. Fahr. (the winter temperature of the tropics); while complete extinction of this pestilence can be counted on, when the temperature falls to freezing-point (Hirsch). Dengue behaves in a somewhat similar way. Cholera and plague behave very differently.

Long-continued residence in a tropical climate is apt to produce in many persons "a slowly-advancing asthenia and cachexia, which almost imperceptibly sap the constitution", due, according to Dr. Birch, to three factors, viz., long-continued high temperature, the presence of malaria, and great diurna variations at certain seasons.

"Their operation is constant, producing excessive cutaneous action, alternating with internal congestions; nor is it possible that any individual can altogether escape them." Degenerative changes ensue, the result of hyperæmia; and the liver, kidneys, and intestines become incompetent. This condition, not so common now as in former years, is distinctly a climatic diseased condition, though not possessing any definite name.

I would submit that climatic disease may be held to include three categories of pathological conditions.

(1) Diseases directly due, either to a single one of the elements of climate, as heat stroke (the microbic origin of siriasis not yet being proved), frost-bite, mountain sickness; or to a concurrence of two elements, as cold and wet, producing bronchitis or rheumatism; or to extreme fluctuation of temperature, causing chill, and so, tropical suppurative hepatitis and (in a partial degree) dysentery.

(2) Specific infectious diseases, which are especially dependent on some meteorological condition, as an important secondary cause; such as, yellow fever and dengue, requiring a high temperature; and (in a partial degree) tubercular phthisis, flourishing mostly in a cold and damp climate.

(3) The deteriorated condition, or cachexia, due to long residence in a tropical climate.

I conclude these introductory remarks with the question, What is Climatic Disease? submitting the above answer for your approval or the reverse. I think that it is not a superfluous question. Among those of us who are called upon to practice abroad there is, perhaps, not always a clear idea of what may, and what may not, be put down to climate. For instance, I have frequently found even enteric fever labelled as climatic. In former days, as Dr. Parkes said, referring to the West Coast of Africa, men were contented to lay their own misdeeds on the climate; the climate was a convenient excuse for pleasurable follies and agreeable vices. We are even now, perhaps, too much inclined to ascribe to this cause the results of sanitary shortsightedness and neglect. But with every limitation it would seem as if there are still some cases in which climate is the essential cause. There are many here this evening who are able to give an authoritative reply to the question, out of the riches of their experience.

Discussion on Lieut.-Colonel Davies's Paper.

Dr. Patrick Manson, C.M.G., expressed himself as generally in sympathy with the paper. There were many instances of the reproduction of so-called tropical diseases in temperate climates, and the recent discovery by Professor Haldane of Ankylostoma Duodenale in the mines of Cornwall was an apt illustration of this fact. The late Dr. Parkes, who was gifted with a remarkable foresight, was clearly of opinion that no very sharp line of demarcation could be drawn in the matter of climatic diseases, and in this sense he (Dr. Parkes) was undoubtedly a prophet. Dr. Manson was not at all convinced that even hepatic disease would for all time remain in the category of climatic diseases, as, perhaps, Col. Davies had suggested. Even at the present time hepatic diseases might, in a sense, be regarded as of parasitic origin, seeing that the alcohol with which the disease was so frequently associated was due to the yeast plant. In his opinion, an altogether undue weight was accorded to cold in relation to a common catarrh. It was clear, he thought, that other factors than cold were absolutely necessary. In the teachings of pathology there was too much "cant," and until this force was discountenanced the science of epidemiology would have an uphill fight before it.

Major R. H. Firth said that it was with some diffidence that he spoke upon this paper, as he feared that he could contribute but little information upon the subject. While being very largely in sympathy with the views enunciated by Col. Davies, he was surprised to find that no reference had been made to the influence of what may be called the physiology of the tropics as a factor in the production of what we call climatic disease. Some very interesting work in this connection was done some years ago by Maurel, Rattray, and Cullimore, and it seemed to him highly desirable that some of the facts should be re-examined by modern methods of research. He fully recognised that we can have climatic influences in the temperate zone as well as in the tropical, but, as his own experiences had been mainly in the latter, he would confine his remarks to the tropical aspect only. There was ample evidence that tropical heat and humidity produced marked changes in body-function, particularly in relation to the processes of metabolism, which exerted an effect adverse to the health and

existence of all but the native-born. These changes were, briefly: an initial elevation in body temperature; a more or less permanent lowering of the pulse-rate and arterial tension, and a lessening of the respiratory function dependent upon deficiency of oxygen intake, due to rarefaction of the air. The net result of these influences exerted a directly deteriorating influence on the blood corpuscles, increased the perspiration, diminished the urinary excretion, and increased secretory action of the liver, whereby some effete matter was passed into the intestines with the bile. But increased liver action meant congestion, and constant congestion involved hepatic deposit, degeneration, and impairment of function. It was in this sense that effects like these were the direct results of meteorological or so-called climatic conditions, that he was of opinion that we must still adhere to the view of the existence of a diseased or abnormal body state, which, for want of a better name, may be called climatic disease. In a tentative way, he had himself endeavoured to elucidate or recognise perverted metabolism among those residing in the tropics. Some two years ago, while serving in the northern Punjab, he kept notes upon the urinary excretion of certain soldiers, more particularly in reference to the elimination of sulphates. As is well known, the urinary sulphates exist in two main forms, the total and the aromatic sulphates, the two groups having in normal conditions a definite ratio. Now, in the men to whom he referred, there were some who showed a persistently high elimination of aromatic sulphates. These latter serve as a fairly reliable index of intestinal putrefaction, and indirectly of imperfect metabolism, especially putrid metabolism, in most cases due to either excessive input of nitrogen or an inability to completely assimilate this class of food stuff. A further curious fact was noticed that all these men who showed this perversion of function succumbed sooner or later to a form of continued fever, known as ardent or simple continued. He was only too conscious of the crudeness of his observations; but he offered them for what they were worth, and as an illustration of what he was compelled to regard as a diseased state directly the result of climatic influences. In this limited sense, we must recognise the existence of direct climatic disease. As for the endemic infective diseases which our forefathers regarded as climatic, we, having a fuller knowledge of their causation, can only admit that they are indirectly climatic, because it is their causative agents only which are directly dependent on climatic states. Yellow fever was a

case in point; limited only to certain climatic areas, not because man can only suffer from it in certain climates, but because the vehicular host, which alone can convey its causative entity to man, can only survive in certain climates. Others, doubtless, would be better able to express alternative views on this really interesting point in epidemiology; but, as an occasionally dweller in the tropics, he could but endorse, in the main, the views expressed by the author of the paper.

LT.-COL. BRUCE SKINNER, R.A.M.C., said that one was reminded by this discussion of what one learned in school-boy classics. We change from square to round, and round to square; what once we loved now we hate; what once we hated now we love. In the present day we know that certain diseases are specific, produced by specific bacteria. These bacteria vary in locality, having a geographical distribution—as all plants and animals have—which depends on local climatic conditions, more or less, as instanced by the Ankylostoma mentioned by Dr. Manson. The old observers taught that we varied with our surroundings, and suffered in consequence. Major Firth had touched upon the variability of our physical functions. He (Col. Skinner) would suggest that Lt.-Col. Davies's paper was interesting as suggesting illustrations of how climatic influences alter our powers of resistance, so that we become (if not perfectly constituted so as to respond to the effects of altered climates on our organs) more liable to fall victims to the attacks of the specific causes of disease, the living germs of which also depend for their preservation on their surroundings.

MAJOR MCCULLOCH, R.A.M.C., said that there was abundant evidence in our military statistical tables to show that residence in tropical climates is commonly accompanied by increased admission-rates for disease, speaking generally, as compared with the admission-rates that obtain at home stations. For example, in the decennial period 1890-1899, the admission-rates per 1,000 of strength among the troops serving in the following parts of the world were:—

United Kingdom	706.5
India	1429.2
China (Hong Kong)	1534.0
West Africa	2972.6

This marked difference in disease-incidence could scarcely be altogether explained by difference in sanitary conditions and surroundings; but part of it, at least, must be due to climatic influences. Further, taking the year 1900, the disease-incidence among the troops in India was greatest among men in their second year of service in the country; it was about equal for men in their first and third years of residence; while, for the later years, the admission-rates show marked decreases. Climatic influences may act by producing local conditions which favour the growth of certain specific causes of disease, and through the lessened resistance of the individual brought about by the systemic disturbance which goes on in the body at first, while the different organs are adapting themselves to the new conditions of life brought about by transference from a temperate to a hot climate.

DR. C. W. DANIELS (London School of Tropical Medicine), said that such tropical diseases as are due purely to climatic conditions will be included in the residuum left after all diseases due to other causes are excluded. Colonel Davies had shown that this residuum is small; but, even in the residuum were included some diseases which could hardly be considered as climatic. Col. Davies instanced hepatic abscess as due, in part at least, to great variations in temperature; but it is common in British Guiana, where the temperature is most equable. With each advance in knowledge, diseases once considered to be climatic "have been proved to be parasitic, and it is highly probable that only an insignificant proportion of the tropical diseases can be correctly attributed to climate."

DR. NEWSHOLME thought that, in the behaviour of certain diseases in this country, we had evidences of a distinct climatic influence. For instance, the factor of meteorological influences could hardly be neglected in a consideration of what is termed the seasonal prevalence of disease; and we had ample illustration of seasonal influences in the behaviour of small-pox, diphtheria, autumnal diarrhœa, etc. Clearly factors, other than meteorological, were operative; but climate or season seemed to him to be one of such factors. In regard to hepatic disease, it would seem, if certain alleged facts were facts, that three factors were operative, *i.e.*, alcohol, climate, and, possibly, *amœba coli*.

Dr. FRANKLIN PARSONS inclined to the view that in the discussion there had been too great a tendency to accentuate the influences of one factor, *i.e.*, climate; whereas, as a matter of fact, in most diseases which had been under consideration, the seed, the soil, and the weather or climate were all operative in some degree.

DR. HARFORD alluded to the fact that no speaker had ventured a definition of the term "climatic," which he regarded as a vague but convenient term, which might be applied in many different ways. The opener had used the term "climatic" as being synonymous with "tropical;" but, if disease were contemplated from the point of view of the tropics, scarlet fever and such-like diseases might be termed climatic.

He considered that the term "tropical medicine" was a convenient one, in the same way as it is useful to class other diseases as medical or surgical for purposes of special study.

Referring to the old idea that a large number of diseases are due to meteorological influences, and especially to chill, whilst it might be difficult to assign another cause, he believed that the number of diseases so produced would be gradually reduced in number, almost to the vanishing point, and that specific causes would be found for most definite diseases.

He quoted Dr. Sambon, as having read an interesting Paper on acclimatisation, in which he seemed to exclude the possibility of any meteorological influences affecting the production of disease. This, Dr. Harford thought, was going too far, and gave it as his opinion that the functions of the body were modified in a change from a cold to a hot climate; as, for instance, in the case of the vascular system, which made the body more ready to become a suitable soil for specific organisms. He noted that disorders of the intestines were peculiarly prevalent in warm climates, which he ascribed largely to the fact that a radical change of diet rendered the bowel peculiarly sensitive to the onslaught of parasitic germs.

THE

BEARING OF OUTBREAKS OF FOOD POISONING UPON THE ETIOLOGY OF EPIDEMIC DIARRHŒA.

By S. DELÉPINE, The Owens College, Manchester.

(*Read: December* 12*th*, 1902.)

Introduction.—It is difficult to avoid some confusion when one has to deal with diseases which it is impossible to define clearly, either by means of *symptoms* and *lesions*, or by means of some *causal agent* capable of demonstration in the majority of cases.

Diarrhœa is a symptom produced by a number of causes, and each one of these causes, according to its *intensity* of action or the *state of the patient*, may produce several types of diarrhœa.

Moreover, there is evidence to show that the agent which initiates an attack of diarrhœa may, in the course of the illness caused by its action, be superseded by other agents (*e.g.*, certain bacteria generally present in the intestine), by which the course of the disease may be so completely altered as to make it difficult to recognise the relations between cause and effect.

In bringing forward certain facts which, in my opinion, establish clearly certain relations between food poisoning and epidemic diarrhœa, I must labour under a double difficulty, for neither food poisoning or epidemic diarrhœa is a well-defined pathological entity.

Definition of Epidemic Diarrhœa.—By epidemic diarrhœa is generally meant *an infectious disease affecting a number of persons at the same time, more especially during the hot seasons*, and one of *the most constant symptoms of which is diarrhœa.* One of its special features is its tendency to occur in certain populous districts, where it causes great mortality among children.

From descriptions I have been able to find in standard books of reference, it appears to me that such terms as *English cholera, cholera nostras, cholera infantum, choleraic or choleriform diarrhœa, septic diarrhœa*, summer

diarrhœa, infantile diarrhœa, etc., must, generally speaking, be taken as synonyms of epidemic diarrhœa. To the morbid anatomist, epidemic diarrhœa is an acute *gastro-enteritis.*

Chief Symptoms.—Epidemic diarrhœa is characterised by a certain number of symptoms, which have been summed up by Dr. Ballard as follows:—"The leading phenomena of the disease are diarrhœa, vomiting, convulsive phenomena; a bodily temperature at certain periods above, at other periods below, what is normal; reduction in quantity or actual suppression of urine, embarassed breathing, indications of pulmonary hyperæmia or inflammation, pallor of surface of the body, loss of bulk and flesh, and exhaustion with its well-known clinical features. Occasionally there is jaundice; now and then a fugitive rash has been observed on the body."

These symptoms agree with those given by various clinical authorities as characteristic of English cholera and infantile diarrhœa, but clinicians include intense thirst, pain and griping, cramps, bilious vomiting and diarrhœa among the important symptoms. Suppression of urine, convulsions and cramps, are not among the characteristic features of the ordinary milder attacks of the disease.

Severe cases of the same disease may present features almost identical with those of Asiatic cholera, poisoning by certain organic and inorganic poisons, and even perforation of the stomach or bowel.

The difficulty of diagnosis of isolated cases is very considerable, and the nature of the disease is generally indicated by its epidemic and seasonal characters, more than by any semeiotic features.

Morbid Anatomy.—In fatal cases the lesions observed are not sufficiently characteristic to remove all doubts. Among these lesions I may mention, absence of food in the stomach and small intestine, hyperæmic, swollen, sometimes ulcerated mucous membrane of the same parts; accumulation of mucus, frequently bile-stained, upon these mucous membranes; degenerative changes in the liver and kidney; hyperæmia of the lungs, sometimes pneumonia; spleen, variable in size, frequently small. There is nothing pathognomonic in any of these lesions.

Bacteriology of Epidemic Diarrhœa.—Exact notions regarding the bacteriology of other forms of enteritis than those connected with cholera, typhoid fever, and anthrax, were very few before 1885, when the classical researches of Escherich upon the *bacillus coli communis* became

known. The researches of that observer showed that although the *bacillus coli communis* is a constant inhabitant of the intestine, it is capable under certain circumstances of acquiring pathogenic properties. Hueppe, in 1887, stated that it was capable of producing cholera nostras, a view which has also been at a later date supported by Gilbert and Girode, and others. Lesage, Macé et Simon, Cumston, and others, have found the bacillus coli communis in a large proportion of the cases of infantile diarrhœa which they have investigated, and believe that organism to be the cause—or at least the most important cause—of summer diarrhœa in children.

Other observers however, have, attributed to other organisms an important share in the production of epidemic diarrhœa.

As far back as 1884, Finckler and Prior found in cases of cholera nostras the *spirillum* which has received their names.

Baginski in Germany, Booker in America, found in the stools and organs of children affected with infantile diarrhœa a number of micro-organisms, among which may be mentioned the *bacillus lactis aerogenes*, the *bacillus coli communis*, the *proteus vulgaris*, *staphylococci*, *streptococci*, etc.

Booker does not believe that the summer diarrhœa of infants is due to any single organism, but nevertheless he attaches some importance to the action of the *proteus vulgaris* and of the *streptococcus enteritidis*. Holst evidently attaches importance to the streptococci, for he has recorded in connection with several epidemics of summer diarrhœa, the presence of the *streptococcus longus* in cow's milk which had been used by the patients. A streptococcus has also been found in connection with acute gastro-enteritis in adults.

Damaschino, Clado, and Lesage, have described a bacillus producing a green pigment, as the cause of a green diarrhœa of infants.

Klein attributes to the *bacillus enteritidis sporogenes*, which he has found in the intestine of children suffering from diarrhœa, an important share in the production of the disease.

This short and incomplete review is sufficient to show that the etiology of epidemic diarrhœa is by no means clear yet. It will be noticed, however, that the bacteria which have been suspected on good grounds of being the cause of outbreaks of summer diarrhœa belong chiefly to

types of bacteria which are inhabitants of the alimentary canal. The virulence of several of these microbes has been shown to be capable of considerable variations.

The bacilli of the colon group appear to be most intimately connected with the epidemic diarrhœa, both of adults and of children. These bacilli resemble each other so closely in some respects, that one is tempted to regard them as varieties of the same species; yet if one submits colon bacilli obtained from the intestinal contents of a number of cases of diarrhœa to the series of tests which are usually employed for diagnostic purposes, one finds that one or more of the reactions which are associated with the *bacillus coli communis* of Escherich are frequently absent. The acid reaction of the culture medium may be permanent, or be replaced more or less rapidly by an alkaline reaction; milk is sometimes coagulated rapidly, sometimes very slowly or not at all; the same variations are observed with regard to the indol reaction, the fermentation of various sugars, the appearances of the growth on potato, and even the serum reaction. The serum reaction presents, with regard to each of the many races of the bacillus coli, such peculiarities of specificity that it is deprived of much of its practical value for the purpose of a clinical diagnosis. In the investigation of limited outbreaks due to the same source of infection, this reaction is, however, very useful for the purpose of determining whether a bacillus isolated from the implicated material, or from some of the cases, is the actual cause of the outbreak.

I cannot, in the time at my disposal, discuss this aspect of the question at greater length here.*

Epidemiological Features of Epidemic Diarrhœa.—The epidemiological features of summer diarrhœa, so well brought out by Ballard's investigation, would appear to be yet the most satisfactory proofs of the specific character of summer diarrhœa.

Food Poisoning.—The resemblance between many outbreaks of food poisoning and epidemic diarrhœa is very great; this resemblance would be even clearer if certain special forms of food infections, such as *botulism*, were clearly separated from the general mass of cases.

Van Ermengem describes, under the name of *botulism*, a state brought about by the ingestion of various articles of food, such as ham, tinned or preserved foods,

* A recent paper by Dr. M. H. Gordon gives a fairly complete account of our present knowledge of the bacteriology of epidemic diarrhœa (*Practitioner*, August, 1902).

oysters, mussels, etc., and which is characterised by comparatively slow onset (twelve to twenty-four hours after infection), secretory troubles, paralysis of certain muscles, dysphagia, constipation, retention of urine, absence of fever, etc. Van Ermengem has found that these symptoms were produced by a bacillus, to which he has given the name of *bacillus botulinus.*

Botulism differs considerably from the more *common form of food poisoning* with which we are more specially acquainted in this country, and which is characterised by practically the same symptoms as those of epidemic diarrhœa.

This last form of food poisoning is, however, very imperfectly understood, as may be seen by consulting our chief books of reference. Observers are generally of the opinion that, in the large majority of cases, food poisoning results from the ingestion of the flesh of an animal suffering from certain forms of septicæmia, enteritis, or pneumo-enteritis, which owes its noxious properties to bacilli, resembling the *bacillus enteritidis* of Gaertner. There is good evidence to show that this occurs at times, but I am of opinion that the mode of infection is usually different and more general.

Works on Hygiene and Public Health attribute the outbreaks of diarrhœa which usually attract attention mostly in connection with hospitals and other public institutions, to the consumption of *water* containing an excess of mineral matters, or contaminated with sewage; of *milk*, which has been exposed to effluvia in ill-ventilated places, or which has undergone fermentation; to *tinned meats, pork pies, ham, game, fish, cheese.* In the latter cases the symptoms are generally supposed to be the results of the action of some chemical poison produced during putrefaction, so that the disease is generally described under the name of *ptomaine poisoning.* Outbreaks of diarrhœa due to the consumption of food are not clinically distinguishable as a group from summer diarrhœa or cholera nostras. Thus Taylor tells us that *acute gastro-enteritis* (*English cholera*), which occurs occasionally during the summer months, is set up by unsuitable ingestia such as sausages, meat pies, shell-fish, or food in a state of decomposition. He also says that there is reason to believe that some of these cases are due to *ptomaine poisoning.* The similarity of the causes of food poisoning and summer diarrhœa is clearly indicated by a great number of clinicians. I exclude from consideration here outbreaks of

typhoid fever due to pollution of food. There are many points of resemblance between food poisoning and food-borne typhoid fever. I think that certain outbreaks of food poisoning have, in the past, been attributed to typhoid fever, and *vice versâ.*

Thus, although we have grown accustomed to designate by different names certain types of diarrhœal disease, it is obvious that these various names do not indicate a reasonable belief in the existence of so many diseases, but relate rather to the *mode of occurrence of a single disease, or of a small group of closely-allied diseases.* General evidence seems to show that the most common forms of food poisoning give rise to symptoms resembling closely those of epidemic diarrhœa. Outbreaks of food poisoning may occur at all times of the year, but they are generally most frequent at the time when epidemic diarrhœa is most prevalent. That they affect adults as well as infants appears to be due chiefly to the kinds of food which give rise to the disease, but certain foods, such as milk, which are partaken of both by infants and adults, give rise in both to attacks of diarrhœa similar to typical attacks of epidemic diarrhœa of unknown origin.

Now, I consider that the chief difficulty which workers have experienced in searching for the actual cause of epidemic diarrhœa, is due to the fact that they have looked for it in the alvine discharges or organs of patients affected with, or who have died from, the disease.

If, as it is reasonable to believe, the bacteria causing epidemic diarrhœa are intestinal bacteria, it is obvious that a great difficulty must be experienced in determining which of these bacteria is responsible for the disease. Many of these bacteria are virulent when introduced into the tissues; many of them increase in virulence in the course of an attack of diarrhœa; and several of them have a tendency to escape from the intestines and penetrate into the various tissues of the body immediately after death, or even during the last hours of life, at the end of an exhausting illness.

On the supposition that epidemic diarrhœa is generally the result of a *more widely disseminated, and less massive form of bacterial infection of food* than is the case with regard to the more definite outbreaks of food poisoning, then we should be able to utilise the occurrence of certain outbreaks of food poisoning for the purpose of turning the difficulties which we have generally to contend with in the investigation of epidemic diarrhœa.

It is with that object that I have for the past eight years investigated the action of bacteria which had given rise to outbreaks of food poisoning, and have also studied certain pathogenic properties of the cows' milk which is supplied to our towns, and which is responsible for much of the infantile mortality due to epidemic diarrhœa. During these eight years I have had opportunities to study outbreaks of infection due to milk, cheese, pork pies, tinned salmon, and other foods. I have also been able to study the pathogenic action of over two thousand samples of milk upon guinea-pigs. By comparing the properties of bacilli obtained from noxious articles of food, and of those obtained from animals which had suffered from the effects of inoculation with various samples of milk, I have been able to satisfy myself that these bacilli, with few exceptions, belonged to the colon group of bacilli.

To show the exact nature of the evidence obtained, I will now give a short account of three of these investigations.

Manchester Outbreak of Diarrhœa, due to the Consumption of Milk, November, 1894.—In the early part of the month of November, 1894, there occurred in Manchester an extensive outbreak of diarrhœa, attributable to the consumption of milk. This outbreak was investigated by Dr. Niven, from whose report I take the following particulars. (Report on the Health of Greater Manchester, 1894, pp. 102, etc.)

On November 7th, Dr. H. Ashby sent a note to Dr. Niven, in reference to a number of cases of illness which had occurred in Victoria Park, Manchester, The symptoms were those usually associated with English cholera. Those attacked were all supplied by one milk-dealer, and Dr. Ashby was of opinion that circumstances pointed to milk being the cause of the outbreak. Dr. Niven ascertained that the attacks in the great majority of instances had occurred on the night of November 5th, and in the early part of November 6th. As a rule, the persons attacked had partaken of unboiled milk. One lady, however, was of opinion that all the milk brought into the house had been boiled. About eight or nine hours elapsed between taking the incriminated milk and the occurrence of symptoms of illness.

Altogether one hundred and sixty cases of illness were reported, occurring in forty-seven families; none of these proved fatal, but in several instances the symptoms were severe.

I need not enter here into administrative matters relating to the steps taken by Dr. Niven to establish the relationship between the outbreak and the milk supplied from one farm. The evidence so collected established clearly the relation.

On visiting the farm, Dr. Niven ascertained that close to the farm-house there was a tip of *midden-privy refuse*, which was estimated to contain 40,000 tons of material of that kind. The farm was bordered by two streams which met below—the one coming from the tip was very foul, the other comparatively clear, but was also contaminated with sewage and with matter from a *tripe-boiling place*. The water used to wash the pails was tepid: its temperature taken by Dr. Martin on one occasion was 92 deg. F. The water used in cleansing the milk pails was kept in a foul cistern. The cows drank from a pool in the yard, which received drainage from the cow-shed midden. The storage of milk over night was such as to expose it to warmth and contamination from the cow-sheds.

Whilst this investigation was proceeding, I was making a bacteriological examination of the only sample of suspected milk which could be obtained on November 8th. I will refer again to this; but it is convenient to state here that without any knowledge of what Dr. Niven was finding, I reported to him that the *bacillus coli communis* which was abundant in the milk, indicated sewage or fæcal pollution, and that it was probable that some contaminated water had found its way into some of the vessels used for collecting or distributing the milk.

Meanwhile, Dr. Niven, on continuing his investigation, discovered a fact which had originally been concealed from him, viz., that on November 8th, a cow affected with inflamed udder (garget) had been removed from the farm, and slaughtered on November 10th.

Several cases of bacterial infection due to the consumption of milk had been recorded in Germany, and in those cases the infection of the milk had been attributed to some disease of the cow.

On this basis, Dr. Niven concluded that the outbreak in Manchester was probably due to the disease of the cow which had been removed from the farm. It is probable that the farmer, on hearing complaints from his customers on November 6th and 7th, got alarmed, and knowing that one of his cows was affected with garget, disposed at once of it, to remove evidence of culpable neglect on his part.

Thus it came to pass that the Victoria Park outbreak

was attributed to some affection of the udder of one cow: a conclusion which had the support of opinions expressed with regard to some previous outbreaks of the same kind.

Results of the Bacteriological Examination of the Milk.—The examination of the milk led me, however, to a different conclusion. The only available sample of the milk which had produced illness was three days old when it reached me; it was firmly clotted, strongly acid, and had an unpleasant sour smell. Six adults or adolescents and an infant, who had partaken of this milk, had been taken ill with violent diarrhœa, sickness, and pain, after a period of incubation lasting about twelve hours.

I gave large doses of this milk to two guinea-pigs and two young rats without producing any distinct illness in those animals.

On microscopical examination, *very few cells* were found, no pus; the fat globules were partly confluent. A very large number of bacteria were found, the most abundant being short, very motile bacilli resembling the bacillus coli, large discrete cocci, and a streptococcus with large segments.* Anaerobic and aerobic cultures in milk and bouillon yielded a very abundant growth of several organisms. I was specially struck with the abundance of bacilli which belonged to the bacillus coli group, Of these there were three varieties—one (A) having all the cultural characters of the typical bacillus coli communis; another (B), which gave all the usual chemical cultural reactions of the bacillus coli communis, but which produced on gelatine thick colonies with rough mammilated surface; a third one (C) had the characters of the bacillus enteritidis of Gaertner.

On testing the virulence of these organisms by intraperitional and subcutaneous inoculation, I found that a single loopful of pure culture on agar of the bacillus (C) produced death within twenty-four hours: the only lesions found after death being a slight amount of peritonitis, intense hyperæmia of the small intestine, congestion of the lungs; there was no enlargement of the spleen.

Pure cultures of the bacillus were obtained from the blood of the heart.

A similar result was obtained by injecting 2 c.c.s. of a pure bouillon culture of the same bacillus under the skin of another guinea-pig.

Bacillus A. proved much less virulent; an abscess formed

* I may say that the streptococcus was not virulent; streptococci are very frequently present in cows' milk having absolutely no noxious properties.

at the seat of inoculation, from which pure cultures of the bacillus coli were obtained.

Bacillus B. did not appear to be virulent.

In the light of the feeding experiments these results at first surprised me, but taking into consideration the state of the milk when it reached the laboratory, it seemed to me possible that the virulence of the milk had diminished as a result of the acid fermentation which had taken place. That the milk had been virulent was sufficiently proved by the effects which it had produced in seven human beings.

Further investigation has shown me that the most noxious among the samples of milk which I have studied, had either an alkaline or an amphoterous reaction.

Fæcal Pollution, the Probable Source of the 1894 *Victoria Park Epidemic.*—The milk which had caused the Victoria Park outbreak contained therefore, among other organisms, a large number of bacilli indicating faecal pollution, rather than infectious disease of the cows. Had the *bacillus enteritidis* been found alone, or associated with only a few typical *coli bacilli,* it might have been said that the bacillus came from one of the milch cows. If this had been the case, it is difficult to understand why the outbreak occurred suddenly. If the noxious milk had been produced by one of the thirteen cows, it is difficult to explain how the infection of the milk was so sudden. In addition to this, the cow which was suspected of having caused the outbreak, was suffering from mastitis, a lesion with which the bacillus enteritidis is not associated.

Non-tuberculous mastitis occurs frequently in milch cows, and I will be able to show that there is no evidence that the milk of cows affected with this lesion is specially noxious.

It seemed to me easier to believe that milk cans or other vessels had been infected on a certain occasion, owing to the use of water polluted with excreta of a *specially virulent kind,* and that the virulent bacteria so introduced had multiplied rapidly in the milk. There was also a distinct possibility of infection through dust, but this seemed to me less probable. The state of things observed by Dr. Niven, on the occasion of his visits to the farm, when they became known to me, confirmed me in my belief in the extraneous origin of the infection: a belief which I communicated to Dr. Niven at the time.

Fæcal Pollution of Milk: A Reasonable Explanation of many cases of Epidemic Diarrhœa.—If, in this case, fæcal pollution of the milk had produced an outbreak of disease

indistinguishable from epidemic diarrhœa, except by its time of occurrence and limited extent, it was reasonable to consider the possibility of a more intimate connection than had been generally recognised between epidemic diarrhœa and fæcal contamination of milk.

Such contamination does frequently occur, as can easily be ascertained by inspection of badly-kept dairy farms, and by the microscopical and bacteriological examination of milk. Under ordinary conditions it is apparently without obvious effects, but when some animals in a herd are affected with intestinal inflammation, virulent bacilli must frequently escape from their bowel and infect more or less directly a portion of the milk, when that fluid is not collected under conditions of strict cleanliness. In summer these bacteria find in milk a suitable medium for rapid growth and multiplication, so that milk originally wholesome may thus be rapidly rendered noxious. On this supposition cows' milk would become most infectious at the time when epidemic diarrhœa is most prevalent, and would affect most severely infants of the age at which cows' milk becomes their chief food, *i.e.*, at the time of weaning.

Working upon that hypothesis, I began an investigation which I have carried on for the last seven years, taking advantage of the large number of samples of milk which were sent to me for the detection of tubercle bacilli.

Those samples were representative of the milk supplied to the inhabitants of large towns. Some were collected at railway stations on their arrival from the country, others were collected at the farms, and represented the mixed milk of a small number of cows; finally, many samples were collected from single—generally diseased—cows. In the latter case, the milk was always collected in sterilised bottles by a veterinary surgeon, who was directed to take every precaution to prevent contamination of the milk by dirt of any kind. By means of these samples, I thought that it would be possible to recognise whether bacilli similar to those which had produced the Victoria Park outbreak were frequently present in the milk supplied to the public. The lesions produced in guinea-pigs inoculated with it, I hoped, would allow me to detect their presence. It would also be easy to recognise the influence of disease of the udder, and of the keeping of milk in warm and cold weather.

Examination of Cows' Milk (as supplied to Towns) for the Detection of Bacteria capable of producing Infection of a Septic Character.—Putting aside a number of preliminary examinations which had to be made for the purpose of de-

termining the course to be followed in the investigation, nearly two thousand five hundred samples of milk have been examined in my laboratory since 1896.

During the years 1896 and 1897 I received many samples from a distance, mostly from Liverpool; these were sent in clean bottles, no precautions being taken to keep the temperature of the milk down. Specimens collected in Manchester were usually brought to the laboratory by hand immediately after collection, either at the railway station or at the farm. As I have already explained, milk from diseased cows was collected by a veterinary surgeon, the cow being milked direct into sterilised bottles which I supplied. Having satisfied myself that milk coming from a distance, without precautions being taken to keep it cool, was infectious in a large proportion of cases, I refused, at the beginning of 1898, to examine any more such specimens unless they were collected in sterilised bottles, and packed in refrigerating boxes which I provided for the purpose. Since that time I have noticed a considerable improvement; and although many of the samples have been sent to me from many distant places including a town over one hundred and ninety miles distant from Manchester, I have had much fewer cases of infection than used to be the case when unrefrigerated milk was sent to me from towns in the neighbourhood of Manchester.

This is shown by the following figures:—

TABLE I.—*Unrefrigerated Milk Examined during the Years* 1896 *and* 1897.

—	Number of Samples.	Number of Samples causing the Death of Two Inoculated Animals in less than Ten Days.			Number of Samples causing the Death of One of the Inoculated Animals in less than Three Days.		
				Per cent.			Per cent.
1896-97	148	5	...	3.3	11	...	7.4
Refrigerated Milk Examined from 1898 *to* 1902.							
1898	111	0	...	0	3	...	2.7
1899	175	1	...	0.57	1	...	0.57
1900	802	4	...	0.50	25	...	3.1
1901	694	1	...	0.14	8	...	1.1

The inference to be drawn from these gross results is clear: a certain proportion of the samples of milk contained bacteria which, under favourable circumstances, gave

to the milk noxious properties, the development of which could be checked in many cases by preventing the growth of these bacteria. The difference between refrigerated and non-refrigerated milk would have been very much greater, *if the milk had invariably been cooled immediately after the milking of the cows.*

The mortality would have been diminished still further in the last three years, if the veterinary surgeons collecting milk at farms distant from Manchester had been able to carry with them refrigerators: owing, however, to the number of specimens they had to collect in a single round, they found it inconvenient to carry a refrigerator about with them; and all that could be done was to bring the samples as rapidly as possible to the laboratory, where they were dealt with without further delay. The rise of mortality which occurred in 1900 was in great part due to the veterinary inspector getting in the habit of keeping a certain number of samples overnight before delivering them to the laboratory: an accident which I did not discover at once.

To find out whether the infectious bacteria present in the milk were chiefly derived from diseased udders or from extraneous contaminations, it is necessary to compare the results obtained with mixed milk generally collected at railway stations, and with the milk obtained from single cows affected with disease of the udder.

The following Table explains itself:—

TABLE II *a.*—*Milk Examined before* 1898. *No precaution being taken to keep the Temperature of the Milk low by Artificial Means.*

—	Mixed Milk Collected at Railway Stations or in Town Dairies.	Milk Collected direct from the Udder of Diseased Cows, the Milk being Collected in Sterilised Vessels.
	Per cent.	Per cent.
Milk producing no noxious effect	37.74	45.76
Milk producing chronic infection (non-tuberculous, not fatal)	37.74	29.12
Milk producing acute infection, rapidly fatal	17.76	0
Milk producing tuberculosis	6.66	24.96
Total	99.90	92.84

TABLE II *b.*—*By the Exclusion of Tuberculous Milk, the following Percentages are obtained.*

—	Mixed Milk.	Unmixed Milk.
	Per cent.	Per cent.
Milk producing no marked noxious effect	40.38	60.5
Milk producing chronic infection, not fatal	40.38	38.5
Milk producing acute infection, rapidly fatal	19.	—
Totals	99.76	99.0

TABLE III *a.*—*Five Hundred Samples of Milk, Examined for the Manchester Corporation during the Year* 1900. *Generally examined within Ten Hours of the time of collection, or refrigerated when kept longer.*

—	Mixed Milk Collected at Railway Stations or Dairies. 357 Samples.	Unmixed Milk of Diseased Cows with Diseased Udders, Collected by Veterinary Inspector. 143 Samples.
	Per cent.	Per cent.
Milk producing no marked noxious effect	67.2	62.2
Milk producing chronic infection, not fatal	18.7	13.9
Milk producing acute affection, rapidly fatal	1.68	1.39
Milk producing tuberculosis	12.3	22.3
Totals	99.88	99.79

TABLE III *b.*—*By Exclusion of Tuberculous Milk there remain* 424 *Samples, giving the following results:*—

—	313 Samples.	111 Samples.
	Per cent.	Per cent.
Milk producing no marked effect	76.6	80.1
Milk producing chronic infection, not fatal	21.4	18.0
Milk producing acute infection, rapidly fatal	1.9	1.8
Totals	99.9	99.9

From these results (Table II*b*) it is obvious that the fatal infection produced in as many as 19.2 per cent. of the animals inoculated with mixed milk could be attributed to disease of the cow's udder in a very few, if any, of the cases. In fact, in this first set of observations, there was not a single case of general rapidly fatal infection produced by milk obtained from diseased udder.

To find out the conditions which favoured the develop-

TABLE IV.—*Mixed Milks coming from a Distance of over Forty Miles, and generally kept for from Twenty-four to Sixty Hours, and even more in a few Cases. (Tuberculous Samples excluded.)*

Mean Temperature in the Shade (Manchester) during Time the Specimens were kept.	Specimens producing no Noxious Effects.	Noxious Specimens.	Totals.	Percentage of Good Specimens.
Deg. Fahr.				
30 to 35	7	5	12	58.0
35 to 40	7	11	18	38.5
40 to 45	2	3	5	40.0
45 to 50	1	4	5	20.0
50 to 55	—	—	—	—
55 to 60	0	2	2	0.0
	17	25	42	39.0

TABLE V.—*Mixed Milks coming from a Short Distance (generally under Twenty Miles), most of them kept for less than Ten Hours (with the exception of Five out of the Seven bad Specimens, and Four out of the Twenty-two good Specimens, which had been kept somewhat longer). (Tuberculous Samples excluded.)*

Mean Temperature in the Shade (Manchester) during Time the Specimens were kept.	Specimens producing no Noxious Effects.	Noxious Specimens.	Totals.	Percentage of Good Specimens.
Deg. Fahr.				
50 to 55	1	0	1	100.0
55 to 60	8	1	9	88 8
60 to 65	11	4	15	73.2
65 to 70	—	—	—	—
70 to 75	2	2	4	50.0
	22	7	29	75.68

TABLE VI.—*Unmixed Milks kept for various lengths of Time, but Collected from the Udder in Sterilised Vessels. (Tuberculous Samples excluded.)*

Mean Temperature in the Shade (Manchester) during Time the Specimens were kept.	Specimens producing no Noxious Effects.	Noxious Specimens.	Totals.	Percentage of Good Specimens.
Deg. Fahr. 35 to 40	6	0	6	100.0
40 to 45	3	2	5	60.0
45 to 50	5	2	7	71 5
50 to 55	—	—	—	—
55 to 60	—	—	—	--
60 to 65	0	3	3	0.0
	14	7	21	67.2

ment of infectious properties, I prepared, in 1896 and 1897, Tables which have already been published as a previous communication ("The Examination of Cows' Milk for the detection of Pathogenic Properties," *The Journal of Comparative Pathology and Therapeutics*, 1897.)

The above short Tables (III, IV, V) summarise the more detailed Tables given in that article, and in which the effects which length of keeping and of temperature have upon the noxious effects of the milk are indicated.

The influence of time is well shown by the number of specimens remaining good, even at a high temperature, when the milk had been kept only half a day. On the other hand, the influence of temperature is still more evident, for in every category the number of good specimens is almost inversely proportional to the height of the temperature. Still, it is important to keep the two factors of time and temperature in mind. *What is produced in a few hours in summer may also occur in winter, when the milk has been kept a long time.*

When the clear relation existing between time of keeping, plus temperature and the noxious properties of a certain number of samples of milk, is contrasted with the ambiguous results obtained when an attempt is made to connect these noxious properties with disease of the udder (tuberculosis being excluded), it is difficult not to feel convinced that infection of the milk outside the udder, and the conditions under which milk is kept, are the

most important factors causing it to acquire infective properties.

I have found a close resemblance between the infection produced in guinea-pigs by the inoculation of milk which had given rise to limited outbreaks of summer diarrhœa, and those produced in animals by the injection of many samples of mixed milks obtained on the market. I believe, therefore, that the presence in the milk of bacilli belonging to the colon group must be the most potent cause of the summer diarrhœa of children. My belief has been strengthened by the fact that, from about 90 per cent. of the cases of fatal septicæmia due to milk inoculation of guinea-pigs, I have been able to isolate bacilli resembling closely, or identical with, those obtained twice from milk causing intense diarrhœa in children and adults. Other organisms have been present in a few specimens, but the only organisms almost constantly present have been these bacilli. These microbes retain their virulent properties even after being cultivated for several generations outside the body. They multiply with extraordinary rapidity at summer's temperature. They resemble the *bacillus coli communis*, or the bacilli of the colon group in their pathogenic action, their mode of growth on gelatine, agar, potato, milk, lactose agar, glucose gelatine, their size, shape, and motility, allowance being made for the variability of the bacilli of this group.

I have not found any reason to alter these views, which I published almost in the same terms in 1897. Since I first expressed, in 1894, my belief in the connection between the infection of food by colon bacilli and summer diarrhœa, Dr. Klein's work on the *bacillus enteritidis sporogenes* has appeared. *Klein's bacillus, which has no relation to the colon group*, would apparently have to milk the same relation as the bacilli of the colon group, being introduced into that fluid by fæcal pollution. All I have said in favour of the view that bacilli of the colon group are essential factors in the production of summer diarrhœa, might be said of the *bacillus enteritidis sporogenes*. The chief difficulty which I find in accepting Dr. Klein's theory is that, out of the large number of samples of milk I have examined by inoculation, I have not found two samples per 1,000 producing lesions which could be attributed to the *bacillus enteritidis sporogenes;* whilst, during the same time, I have found that from ten to fifty or more per 1,000 (according to the season or length of keeping) were capable of causing infection attributable to a bacillus of the colon group.

Streptococci did not seem to be responsible for a greater proportion of cases than the *bacillus enteritidis sporogenes.**

Comparison of Epidemiological data, and of Bacteriological data resulting from the Examination of Cows' Milk.—A consideration of the facts brought to light by the valuable contributions of Dr. Ogle, Dr. Ballard, Dr. Newsholme, and others, impresses me with the belief that temperature is, on the whole, the most important of the factors determining the rate of mortality from epidemic diarrhœa.

Dr. Newsholme's able and exhaustive "Contribution to the Study of Epidemic Diarrhœa" (*Public Health*, vol. x, p. 139, 1899–1900), leaves no doubt upon this subject. The influence of other conditions is fully discussed in Dr. Newsholme's address; but, although those conditions have undoubtedly to be considered, they are, in my opinion, of secondary importance with regard to their relation to case-incidence.

Dr. Newsholme concludes that the disease is due to the infection of the air by a particulate poison which infects the air, and is swallowed, most commonly with food, especially milk. In this he agrees with Ballard, and with regard to milk he is also in agreement with the position I had previously taken.

In discussing the influence of milk, however, Dr. Newsholme does not attach so much importance as I had to infection at the cow-shed. He is of opinion that milk is probably generally infected during storage at home, in places where it is exposed to pollution by infective dust.

My results do not exclude infection at the home of the consumer, or during transit from the farm, but they indicate that infection at the farm, or through vessels infected at the farm, and used by the farmer for the storage and carriage of milk, must be of paramount importance. It will be noticed that a large proportion of the samples of milk obtained from cans at railway stations, or at the farms, *are infectious before the milk reaches the consumer.* The degree of noxiousness it acquires through that infection is proportional to the length of time the milk

* It is somewhat difficult to estimate the share taken by streptococci and staphylococci in the production of lesions following milk inoculations; they are found frequently enough in animals dying more than ten days after inoculation, and they may in such cases be associated with bacilli of the colon group, but they are not usually found in animals dying from acute septicæmia. It is probable that, in many instances, their presence is due to secondary infection.

has been kept, and the temperature which it has been exposed to before it reaches the consumer. It is, therefore, obvious that long keeping and high temperature, are the two important factors which determine whether a sample of infected milk will contain a sufficient quantity of bacteria or bacterial products to produce infection. The conditions necessary for this development of noxious properties are generally more easily attained during transit from the farm in hot railway vans than after the milk has reached the home of the consumer. The same factors are, of course, at work at the last place, but usually the time is shorter and the temperature lower. Infection, long keeping, and high temperature, are all compatible with many town cellars or pantries; but, taking all the facts I have collected into consideration, it appears to me that infection at the farm, or through milk cans or other vessels, is by far the most important factor. According to this view, the explanation of infantile diarrhœa would be an easy matter; infection through cows' milk explains readily the special incidence of the disease and the high mortality of infants between the third and the twelfth month. The difficulty of obtaining fresh milk in the centre of populous districts would also explain why so much of the milk consumed by children of large towns is infectious, for such milk has often to travel over considerable distances under very unfavourable conditions. The better classes are generally supplied with fresher milk than the poorer classes. Condensed milk, which is so much used by the latter, is not, as is often believed, sterilised. It often contains a large number of bacteria and much dirt. Finally, the rapidly-multiplying bacillus when once introduced into a feeding-bottle, which is not frequently sterilised, will render the bottle itself a source of infection, and any milk subsequently placed in the bottle. Such an accident is more likely to occur in homes which are unclean, where the mother who works out of doors has to leave her last born to the care of a previously-born child, or to strangers.

The epidemic diarrhœa of adults is not so easily explained, because milk does not form such an important part of the regular food of the adult population; and also because the adult is much less liable to suffer from the consumption of contaminated food. It has, however, to be remembered that milk is not the only food which is exposed to fæcal contamination: meat, fish, molluscs, vegetables, fruit (fresh or preserved), are all liable to pollu-

tion, especially when prepared for consumption on dirty premises.

The study of outbreaks like the one I have recently investigated in Derby, has convinced me that specific fæcal pollution, generally at the time of manufacture, of various articles of food, is responsible for a large number of epidemics of food poisoning.

Some Remarks about the Derby Outbreak of Food Poisoning.—The pork pies which caused the Derby outbreak contained a large number of bacilli having characters closely allied to those of the *bacillus enteritidis* of Gaertner, and of various bacilli which I had isolated from pathogenic milk during the last eight years. A careful examination of the pies led me to the conclusion that the meat of those pies had been infected by coming in contact with some infectious fæcal matter. An inspection of the premises where the pies had been prepared convinced me that such an infection was not only possible but probable, and I was even able to indicate the part of the premises where the infection had occurred. My conclusions were entirely confirmed by subsequent inquiries conducted by Dr. Howarth, who found that the meat which had been used in the preparation of the most noxious batch of pies had been exposed to special fæcal pollution, owing to the cleaning of apparently diseased bowels in a room where the pie meat was left in uncovered vessels.

A specially interesting feature of the Derby outbreak is, that although a large number of persons (at least one hundred and thirty) fell ill in Derby, none died during the epidemic. (One fatal case occurred a considerable time afterwards, from complications, which were possibly, though not necessarily, the result of the infection.)

Of the cases (about ninety) occurring outside the borough, some at a considerable distance, at least four ended fatally. (I was able to prove the presence of the *bacillus Derbiensis* in the organs of two of these fatal cases; I had not an opportunity to examine the organs of the others.)

Thus the pies had increased in virulence on being kept, just as milk does when it is sent from a distance to a town, and is not consumed soon after collection.

Is there a Specific Bacillus capable of causing Epidemic Diarrhœa?—I have previously stated that, from the milk which caused the Victoria Park epidemic, I isolated several bacilli belonging to the colon group, one of which was very virulent, causing rapid septicæmia when injected into

the peritoneum, and somewhat less rapid septicæmia when injected under the skin of guinea-pigs. The chief lesions observed after death were congestion of the small intestine, which contained only mucus (more or less bile-stained), great congestion of the lungs; the spleen was small, or slightly enlarged. From the blood of the heart and of all the organs the bacillus could easily be recovered. This bacillus had characters resembling closely those of the *bacillus enteritidis* of Gaertner. The lesions observed in about 90 per cent. of the guinea-pigs which succumbed rapidly to the inoculation of milk supplied to towns, were similar to the above: with that difference that in a few cases the spleen was more enlarged, or that peritonitis was more marked. The bacillus isolated from the blood of these animals resembled more or less closely the bacilli obtained from the milk which had caused the Victoria Park epidemic. In four cases out of ten, a pure culture of a bacillus, resembling the *bacillus enteritidis* of Gaertner, was obtained; in the other cases I found a mixture of bacilli belonging to the colon group, some approaching the type of Gaertner's bacillus, others resembling more the *bacillus coli* of Escherich.

Animals fed on the pies which had caused the Derby epidemic, presented after death lesions similar to those which I have described above, and the bacillus isolated from their blood had characters allied to those of the *bacillus enteritidis* of Gaertner.

From the blood of two patients who had died from the effects of the pies, the same bacillus as that obtained from the organs of guinea-pigs fed on the pies was isolated, but it was accompanied by bacilli identical with or resembling the *bacillus coli communis.*

All these facts put together, as well as many of those which have been previously recorded, indicate that the infectious properties which food acquires frequently in summer, and which give rise to the ordinary type of epidemic diarrhœa, are due to bacilli belonging to the colon group of bacilli, of which the *bacillus coli communis* of Escherich and the *bacillus enteritidis* of Gaertner are the two extreme types. I have come also to the conclusion that the varieties of those bacilli which are the most important sources of infection are those which resemble the bacillus of Gaertner, and which, therefore, produce *no permanent acidity, coagulation, or distinct smell when grown in milk.* Very few of the *infectious samples* of milk which I have examined during the last five years had a

distinct acid reaction, so that *absence of acidity in milk is not, as generally believed, an index of safety.*

It is probable that the most dangerous kind of fæcal infection is that produced by matter containing bacilli resembling Gaertner's bacillus. *Such an infection is possibly connected with the existence of an infectious diarrhœal disease liable to occur in the lower animals as well as in man.* I have not, however, been able to satisfy myself regarding that point.

It is certain, however, that bacilli presenting the characters of the ordinary *bacillus coli communis* are seldom capable of producing such a rapid infection as that produced by the *bacillus enteritidis*, or by closely-allied bacilli such as the *bacillus enteritidis Derbiensis.* I hope that an investigation which I am at present conducting, with the co-operation of Dr. A. Sellers, will allow me to speak before long more definitely with regard to the properties of this group of bacilli. I wish to acknowledge here the great help which I have obtained from various colleagues in the collection of data necessary to the interpretation of my experimental results. I wish specially to thank Dr. Niven for the assistance he has so willingly given me in that respect.

General Conclusions.

1. Epidemic diarrhœa of the common type occurring in this country is apparently, in the great majority of instances, the result of infection of food by bacilli belonging to the colon group of bacilli, and which are derived from fæcal matter.

2. It appears that this infection of food does not generally lead to obvious consequences, unless the infection is massive or the food is kept for a length of time, and under conditions of temperature favouring the multiplication of these bacilli.

3. Milk, which is the most common carrier of the cause of epidemic diarrhœa in infants, is very frequently infected at the farm, or (through vessels) in transit. It may also be infected at the retailer's, or at the consumer's house.

4. Of the bacilli of the colon group which are capable of rendering the milk infectious, those which do not produce a large amount of acid, and do not coagulate milk, are the most virulent, and are probably the essential cause of epidemic diarrhœa.

Preventive Measures against Epidemic Diarrhœa.—The preventive measures which seem to me to be most clearly indicated by the above conclusions are the following :—

1. Measures securing *cleanness* of cows, dairy hands, cow-sheds, milk vessels, etc. Similar measures are also needed in other places, with regard to all things or persons coming in contact with milk or any other article of food, manufactured or not.

This, I think, is generally admitted to be a most important element of good sanitation; but absolute cleanness is most difficult to obtain, if not practically impossible. Occasional infection must, therefore, occur now and again.

2. To guard against the effects of accidental fæcal infection, the food should be *consumed fresh,* when possible.

3. When the food cannot be consumed fresh it should be *refrigerated, i.e.,* kept at a temperature below 4 deg. Cent.

4. Where the food cannot be eaten fresh or refrigerated, it should be *sterilised* by heat (*i.e.,* by thorough cooking).

(If I were certain that the noxious bacilli are derived only from animals affected with enteritis, I would suggest isolation of such animals as an essential measure, even though I believe that such a measure would be extremely difficult to carry out).

N.B.—The above precautions refer only to epidemic diarrhœa; other forms of infection may be dealt with in other ways; thus the only safeguard against tuberculous milk is boiling of the milk, or entire exclusion of tuberculous cattle from our herds.

APPENDIX.

The use of preservatives is often a source of danger. I have made a number of experiments on the effects of borax and boracic acid upon the bacillus of typhoid fever, bacillus coli communis, and the bacillus enteritidis in milk, and found that these organisms continued to multiply in milk at summer's temperature, in the presence of 35 grains, 70 grains, and 140 grains of the usual mixture of boracic acid and borax per gallon of milk. The same quantities of the preservative delayed the appearance of the acid reaction from one to two days, and the clotting from one to three days. The milk had, therefore, time to become highly infectious before its character had become suspicious. Therefore, the use of preservatives, by allowing a *stale article to be sold as a fresh article,* is a source of danger.

REMARKS ON THE CAUSATION OF EPIDEMIC DIARRHŒA, INTRODUCING THE DISCUSSION ON PROFESSOR DELÉPINE'S PAPER.

By ARTHUR NEWSHOLME, M.D.

(*Read: January 16th*, 1903.)

Professor Delépine's important paper goes far towards laying the foundation for a more complete knowledge of epidemic diarrhœa than we have hitherto possessed, and thus renders possible active preventive measures against this serious disease. The causation of this disease has been attacked by him from a different standpoint to any hitherto taken. Instead of searching directly for the active cause of epidemic diarrhœa in the alvine discharges or organs of patients affected with the disease, he investigates (*a*) outbreaks of poisoning by solid foods, like the Derby pork-pie epidemic; and (*b*) the pathogenic properties of milk as supplied to Manchester. Under the first head, he brings forward convincing evidence that epidemics of meat poisoning are commonly due to fæcal contamination, in which a more massive form of bacterial infection of food occurs than when milk is contaminated; but that in each instance organisms of the *B. coli* group are the pathogenic agents, especially those closely allied to the Gaertner bacillus. Under the second head, he shows that a large percentage of samples of fresh milk supplied to Manchester are pathogenic when inoculated in guinea pigs, and that this pathogenicity is greatest when the milk has travelled a long distance, and when it has been subjected to summer temperatures; also, that usually a virulent bacillus of the coli group can be recovered from the guinea pigs dying from the acute septicæmia caused by inoculation of these pathogenic milks. Such are the important facts brought out by Professor Delépine's work, which embodies the result of seven years' observations and experiments, and is therefore entitled to the greatest weight. He proves, furthermore, that milk is seldom pathogenic when obtained from the individual cow; and he concludes that it is infected

chiefly on the farm or (through vessels) in transit. He suggests that enteritis in the cow may be the source from which virulent bacilli are derived, and subsequently infect the milk. If this suggestion prove correct, the veterinary aspect of enteritis will become important; and improved means of diagnosis of this disease, followed by isolation of affected cows, will be as important as attention to strict cleanliness in connection with the operations of milking, cooling, transit, and storing of milk.

One link is missing in the chain of evidence connecting infection by coli bacilli with the causation of epidemic diarrhœa. The serum reaction of the blood of patients suffering infection by the colon-typhoid group of bacilli gives some indication of the particular bacillus by which the infection was caused. Professor Delépine only briefly refers to this aspect of the investigation; it would be interesting to know whether the blood of children suffering from diarrhœa after drinking milk from the same source as the milk found by him to be pathogenic was examined in any instance, and their serum reaction compared with that given by blood-serum from the affected guinea pigs, and with that given by known varieties of the Gaertner and other bacilli of the coli group. I venture to express the hope that during the coming summer this line of inquiry will be pursued by many investigators; and that ere long, by a continuance of Professor Delépine's work and by work on these lines, we shall be in a position to state whether epidemic diarrhœa is caused by one particular *B. coli* or by several varieties. Lesage* appears to have made some investigation on the lines indicated above. He found that the *B. coli* coming from a child in the acute stage of epidemic diarrhœa was agglutinated by the serum of the same child. This fact he verified in 40 out of 50 cases of the disease. In these 40 cases the serum of each child agglutinated also thirty-nine examples of *B. coli* from thirty-nine other children seized with the same affection. The reaction disappeared in a few days. According to him, the *B. coli* of the normal intestine of infants is not agglutinated by the sera of the affected children, and the normal serum does not agglutinate the infecting *B. coli* nor the normal *B. coli*. It is most important that these results should be confirmed and extended.†

* Quoted by Dr. M. H. Gordon, *Practitioner*, August, 1902, p. 183.

† As opposed to the practicability of researches on the lines pursued by Lesage, the following remarks by Drs. Lorrain Smith and J. Tennant (*British Medical Journal*, December 27th, 1902, p. 1941), may be quoted:—

Meanwhile I venture to express agreement with Professor Delépine's important induction that "epidemic diarrhœa is apparently, in the great majority of instances, the result of infection of food by bacilli belonging to the colon group of bacilli, and which are derived from fæcal matter." It is with a sigh of relief that I do so, for in December, 1899, I expressed the hope that the *Bacillus enteritidis sporogenes*, suggested by Klein to be the cause of epidemic diarrhœa, might not be so, as this particular micro-organism possessed a very persistent vitality, and milk could scarcely be freed from it without spoiling it as a food.*

But, while agreeing with Professor Delépine that epidemic diarrhœa is caused by fæcal infection by *B. coli*, I must venture to dispute his conclusion that milk "is usually infected at the farm or in transit." The points which favour Professor Delépine's view are, I think, as follows:—

(1) There is the analogy of the Derby pork-pie cases. No deaths occurred in Derby itself; several among those who ate Derby pork-pies at a distance, presumably after a longer time had elapsed, enabling a greater multiplication of coli organisms to occur. (2) There are the facts, so well brought out by Professor Delépine, that the percentage of pathogenic samples of milk was greatest when milk had been brought from long distances and kept a long time. (3) Fæcal contamination of milk appears to be more likely to occur on the farm than at any later stage before it is used as infants' food.

Are we certain, however, that the infection is conveyed by milk in such a preponderant proportion of cases as is commonly supposed to hold good? Milk is undoubtedly a vehicle of diarrhœal infection, probably the most common vehicle. On this point I cannot do better than quote Professor Delépine: "in discussing the influence of milk . . . . Dr. Newsholme does not attach so much importance as I had to infection at the cowshed. He is of opinion that milk is generally affected during storage at home, in

"It often happens that even when a race (of *B. coli*) which does not easily agglutinate is used for inoculation, the resulting serum willgive a reaction with other races of *B. coli* at a dilution much higher than the maximum dilution which will agglutinate the inoculating race. It is consequently impossible to use the serum reaction of the *coli* group of bacilli in the same definite way as may be permitted with the typhoid group, in which the tendency to agglutinate is much more uniform. There is, consequently, an element of uncertainty in interpreting the meaning of *coli* reaction in a given instance." A similar conclusion is stated by Professor Delépine, in his Paper.

* "A Contribution to the Study of Epidemic Diarrhœa," *Public Health*, December, 1899, p. 13.

places where it is exposed to pollution by infective dust." In order to discuss this point, I propose to give briefly the results of an investigation into the milk supply of the 226 fatal cases of diarrhœa which occurred in Brighton in the three years, 1900-02. In 35 of these cases the milk supply could not be ascertained, or (in 4 of the 35) was derived from mixed sources. There remain, therefore, 191 cases of known milk supply. Of these 18, or 9.4 per cent., were breast-fed, and 84, or 44 per cent., were fed on condensed milk. Thus in over 53 per cent. of the cases of epidemic diarrhœa in Brighton with known milk supply, domestic infection, so far as milk is concerned. was alone possible. Great care was taken to exclude from these groups cases in which other sources of milk than the breast or condensed milk were on any occasion employed. It is clear, therefore, that in Brighton, *even on the assumption that the contagion of diarrhœa is always conveyed by milk,* domestic infection of milk can alone be responsible for more than half the cases. I assume that condensed milk is not, except in rare instances, infective when the can containing it is first opened. We know that mother's milk is sterile, and that any contagion conveyed with it must be derived from a non-sterile nipple; and yet over 9 per cent. of the total cases of diarrhœa were among breast-fed infants. In Liverpool, of 1,096 deaths from diarrhœa in the third quarter of 1899, 225, or 20.5 per cent., were breast-fed.* These facts do not prove that fresh cow's milk is not a more frequent vehicle for the contagion of epidemic diarrhœa than breast milk or condensed milk; nor do they disprove it. To arrive at a definite conclusion on this point, we need to know the proportional number of infants suckled, fed on condensed milk, and by fresh cows' milk, respectively. No such figures are, so far as I know, available. Meanwhile, one can only surmise. Unless we assume that more than 44 per cent. of infants in Brighton are fed on condensed milk, we are driven to the conclusion that, if milk has any relationship to diarrhœa, condensed milk favours diarrhœa. I am inclined to think that owing to its greater digestibility, and notwithstanding the fact that a tin of condensed milk after being opened may not be finished for several days, condensed milk is less prone to produce diarrhœa than fresh cows' milk. It suffices, however, for my present argument, to have shown that fæcal contamination at the farm, in the cowshed, and

* Dr. Hope's Report; quoted by Dr. Waldo, *Lancet*, May 14th, 1900, p. 1427.

during transit by road or rail, cannot possibly have been the cause of more than 47 per cent. of the total fatal diarrhœa in Brighton during the last three years, unless it can be shown that condensed milk is commonly infective before the tin is opened. I do not propose to press the argument further; but I may ask whether, in view of the fact that in half the total cases of diarrhœa, infection from the cow and its environment is rigidly excluded, it is not possible that in a considerable proportion of the remaining half, in which fresh cows' milk was taken, this was not the vehicle of infection?

Professor Delépine has conclusively proved that milk is occasionally the cause of serious epidemics of diarrhœa, as in the Victoria Park outbreak, described by him; and he has shown that fæcal contamination of milk is the cause of these epidemics. Such outbreaks are analogous to milk outbreaks of enteric fever. These always occur on a large scale, only limited by the number of susceptible persons supplied with milk from the implicated supply. Sporadic cases of enteric fever in connection with milk are almost, if not quite, unknown. Cases of meat-poisoning—the pathological relationship, almost amounting to identity, between which and epidemic diarrhœa has been indicated by Professor Delépine—occur in epidemics, the size of which is only limited by the number who have partaken of the infected food. We can, therefore, have no hesitation in agreeing that specific infection of milk at the farm, or in transit, is responsible for those epidemics of diarrhœa occurring in connection with a particular milk supply, just as in the analogous case of a milk epidemic of enteric fever. The balance of evidence still, I think, leans to the conclusion that the ordinary sporadic cases of diarrhœa are due to domestic infection of milk or other foods, or to the direct swallowing of infective dust. The statistics of milk supply in Brighton throws some light on this point. The following Table shows the different supplies of milk among the families invaded by diarrhœa during the last three years:—

Source of Milk.	Number of Deaths from Diarrhœa.
Condensed milk	84
Human milk	18
Sterilised milk	1
Dairy A	13
Dairy B	6
Dairy C	5
Dairies D, E, F, G, H	4 each
Dairies I, J, K, L	3 each
Dairies M, N, O, P, Q, R	2 each
Twenty other Dairies	1 each
Thirty-one other Dairies	No deaths

Thus, with the possible exception of Dairy A, which supplies a large portion of a very poor district, there is no evidence of special incidence on single milk supplies. I next investigated the sources of milk supply for the whole of Brighton, in accordance with the facts as to whether (*a*) any fatal cases of diarrhœa had occurred in connection with them ; and (*b*) whether the milk was brought in by road or rail. This point is important, as milk brought in by road comes from neighbouring farms, and is more likely to be newer than rail-borne milk. No attempt has been made to classify the dairies according to the amount of milk they supply (or, what would be more important, the number of families they supply—in a poor district a given sale of milk would mean a supply to three or four times the number of families served in a neighbourhood of higher social status). It is, however, the fact that the dairies supplied by rail are, as a rule, larger than those supplied by road. The following Table summarises the results of this inquiry :—

Milk.

—	A. Road-borne.	B. Rail-borne.	C. Parts of Supply Road- and Rail-borne.
Number of dairies having no diarrhœa among their customers	8	21	2
Number of dairies having diarrhœa among their customers	10 (22 cases)	26 (57 cases)	2 (9 cases)

It will be seen from the above Table that, whether the number of dairies implicated, or the number of cases of diarrhœa occurring among the customers of these dairies be taken, the percentage of diarrhœa is equal—whether the milk is road-borne or rail-borne.

Combining the results of the first and second Tables, we notice that during the three years under consideration thirty-one dairies had no fatal cases of diarrhœa among their customers, twenty dairies had one fatal case each, and eighteen had larger numbers of cases, the largest number in connection with any one dairy being 13 cases in three years. Unless we assume that a very large proportion of non-fatal cases occurred, we must infer that

most of these milk supplies were, as regards diarrhœa, non-infective before they reached the home of the individual patient.

In view of the preceding considerations, I still retain my opinion that domestic infection is the most common source of diarrhœa. One might with advantage, did time permit, enlarge on the analogy between epidemic diarrhœa and enteric fever, which has been already indicated. In both the excreta are infective; in both, personal infection almost certainly bears a much larger share in the spread of the disease than is generally recognised; in both, so far as our English experience is concerned, the conditions producing sporadic cases are in the aggregate a more extensive cause of disease than the conditions producing water or milk epidemics. Multiple cases of diarrhœa in households are not uncommon; flies convey infection from one house to another. Those who frequently visit the homes of the urban poor in summer will realise that food in them can scarcely escape fæcal infection. There are no pantries; food is stored in cupboards in a living room or bedroom. The sugar used in sweetening the baby's milk is often black with flies, which have come from a neighbouring privy or manure-heap, or even from the liquid stools of a diarrhœal patient in a neighbouring house. Flies have to be picked out of the half-empty can of condensed milk, before its remaining contents can be used for the next meal of the infant. Hence, while agreeing with Professor Delépine's conclusion that fæcal contamination of food is responsible for most of our epidemic diarrhœa, I conclude that the infection is oftener domestic than bucolic in origin. The foul condition of feeding-bottles, whatever the source of milk, gives ample time as well as a suitable temperature for the multiplication of coli bacilli in the intervals of feeding of infants. These coli bacilli may sometimes originate from the cow; they may also, as Dr. Waldo has suggested, be derived from horse-manure. We cannot afford to neglect either of these possibilities; but in my opinion human coli bacilli are probably the most common source of infection; and in this disease, as in tuberculosis, our chief enemy is in our own and in neighbouring households. When we remember the habits of personal uncleanliness of a large share of the population; when we recollect the fact that the mother preparing her infant's food commonly does so with unwashed hands, the inoculation of this food with virulent coli bacilli of human origin ceases to be a matter of surprise. If my contention be correct, the following among other

reforms are required before we can hope for much reduction in diarrhœal mortality:—

(1) Exact instruction, as suggested by Dr. Niven, of older girls in our day schools in the art of preparing infants' food; in the principles and practice of sterilisation of food and of bottles; and in the minutest cleanliness in connection with the storing and handling of food.

(2) Greater domestic and municipal cleanliness: involving as this will the reduction of dust both in houses, courts, and streets; and the substitution, both domestically and municipally, of wet cleansing and more frequent cleansing for the dry and infrequent cleansing now in vogue.

(3) A crusade against the domestic fly, which is most numerous at the season and in the years when epidemic diarrhœa is most prevalent, and probably plays a large part, as already indicated, in spreading infection.

The subject of flies is closely related to the consideration of the climatic conditions known to favour epidemic diarrhœa. I discussed these in detail, in a former paper,* and can now only make two or three summary remarks. If it be true, as Professor Delépine has shown, that coli bacilli are the cause of epidemic diarrhœa, and if, as I have contended, direct infection from patient to patient, or indirect infection by flies and dust, is chiefly responsible for the spread of this disease, what bearing have these facts on the climatic condition known to favour epidemic diarrhœa? The two climatic conditions which have been shown specially to influence diarrhœa are rainfall and temperature. Thus, "given two towns equally placed, so far as social and sanitary conditions are concerned, their relative diarrhœal mortality is proportional to the height of the temperature and the deficiency of rainfall of each town, particularly the temperature and rainfall of the third quarter of the year"* On the basis of figures given in the paper from which the above remark is quoted, I have stated that "this relationship is so close, that the towns may be classified meteorologically in the order in which they ought to stand in reference to diarrhœa; and their true relative position as to domestic and municipal sanitation may be ascertained when we know whether they occupy a better or worse position on the list of towns than that which their meteorological place would indicate as rightly belonging to them." A careful examination of available data has satisfied me that the incidence

* *Public Health*, December, 1899.

of diarrhœa follows more closely the rainfall than the mean temperature of the air. On the hypothesis of coli infection, this might have been anticipated. Frequent rains mean frequent removal of infective material, and they probably also prevent the movements, if not the actual hatching, of the domestic fly. Exact observations on the breeding-places, the duration of hatching, and the conditions generally favourable or inimical to the multiplication of the fly, are still a desideratum.

One word in conclusion on the relation between soil—temperature, and diarrhœa. Earth temperatures at a depth of 4 ft. have become widely accepted as an important index of the possibility of prevalence of epidemic diarrhœa; and it has been accepted almost as an axiom that we must expect diarrhœa to prevail when the 4-ft. earth thermometer has reached Ballard's critical point of 56 deg. Fahr. The experience of last summer shows the fallaciousness of this indication. In Brighton the 4-ft. earth thermometer reached 56 deg. Fahr. on the 24th June. On the 30th June it was 58.4 deg. Fahr. During the second half of July it remained between 60 and 61 deg. Fahr. In August it reached 61.8 deg. Fahr. It reached its maximum of 62.6 deg. Fahr. on the 10th September, then slowly falling, but keeping above 56 deg. Fahr. until the 19th of October, when it was 55.8 deg. Fahr. But no deaths from diarrhœa occurred in the weeks ending with Saturday, July 5th. In the six following weeks only 3 deaths from diarrhœa occurred; and, contrary to Ballard's rule, the highest number of deaths from diarrhœa in any one week did not occur in the week in which the 4-ft. earth thermometer attained its mean weekly maximum, but three weeks later. Similarly in Croydon—for information concerning which I am indebted to Dr. Meredith Richards—no deaths from diarrhœa occurred in the five weeks ending August 2nd, the highest 4 ft. earth thermometer during this time ranging from 56.8 deg. Fahr. to 59 deg. Fahr.; while the weeks of maximum number of deaths from diarrhœa coincided in this town with a second maximum of the earth temperature, which was lower than the maximum 59 deg. Fahr. in the week ending July 26th, when no deaths from diarrhœa occurred. The key to the anomaly is found in the fact that in each week of the third quarter of 1902 more or less rainfall occurred. The temperature conditions were favourable to diarrhœa, the rainfall prevented it. The facts for the present year justify my conclusion that rainfall is more important than temperature in relation to

epidemic diarrhœa. Hence the excellent records of Huddersfield and Halifax. If Oldham, and in a much higher degree Liverpool, do not stand so well as their rainfall records would lead one to expect, it is in spite of nature's help, and because there are social and sanitary conditions tending to counteract the beneficent influence of nature's operations.

Discussion on Professor Delépine's Paper.

Dr. Meredith Richards thanked Professor Delépine for his interesting paper. The first two or three years of his work as Medical Officer of Health at Croydon brought him face to face with this problem—epidemic diarrhœa. The rate went up by leaps and bounds. He should not like to dissent from Professor Delépine's conclusion that the cow is an important factor. He wished it were of chief importance: it would then be necessary to attack dairies only. He would ask if diarrhœa was due to *coli* infection occurring at farms? If so, it would be worse on hot days. His next point: if infection took place only at the farm, why should wet weather have the very important effect of checking diarrhœa? Can we think the wet weather lowers the air temperature and checks development of *coli*? For some years he had made observations of diarrhœa mortality. At Chesterfield he found 11 per cent. occurred among children fed from the breast. Of those surviving—*i.e.*, at the end of six months—42 per cent. were fed from the breast. At another time, 240 deaths from diarrhœa occurred at Croydon, a percentage almost the same as Dr. Newsholme's: 12 per cent. were fed from the breast, 40 per cent. from manufactured food—mostly Nestle's—the rest were cow-fed. At another time he had a record of 36 deaths, of which 66 per cent. were receiving cow's milk. If these figures are correct, he would suggest that infection takes place at the home and not necessarily at the cow-shed. He was particularly interested in conclusion No. 5. He had been trying to get authority to obtain powers to fix a standard of pureness for milk. To get powers (1) to prevent the exposure of milk for sale with more than a certain proportion of alkali; (2) To prevent the selling of milk not properly cooled; (3) To stop the selling of acid milk. His idea was that possibly acid milk might account for epidemic diarrhœa. Dr. Delépine thought it was not

so. Could it be used a few hours after it had been bought? He should like to ask if it was practicable to do what has been done in New York: to make it illegal to offer for sale in summer milk with more than a certain proportion of bacilli. Apparently, milk is not particularly virulent in spring and autumn. Lastly, he might say that he had analysed dairy supplies, and on the whole his results were the same as Dr. Newsholme's. He was sorry to say that he could not find that milk from a distance was worse than local milk.

DR. NASH said that all must admit the coincidence of the earth temperature and the diarrhœa mortality, and no doubt an indirect causal relation obtains; but he joined issue with Dr. Ballard in his conclusions that the essential cause of diarrhœa is a specific *micro-organism* residing in the superficial layer of the earth, whose vital manifestations are dependent upon conditions of season, etc. Dr. Nash was prepared to admit that the essential cause is bacterial in nature, but he thought that all evidence went to show that there *is no one specific bacterium* of diarrhœa. If instead of a micro-organism we merely said an "organism whose vital manifestations are dependent upon conditions of season," we should, he thought, be nearer the truth. He suggested that this organism is the common house-fly, which, beginning to make its appearance in June, becomes a veritable pest during July and the early part of August. Towards the end of August its existence tends to become a mere sexual one, and it rapidly decreases in numbers thereafter, though occasional specimens may be found into November. This creature is often termed the harmless fly. He certainly gave it the first place as a pathogenic agent during the summer months. It is a useful scavenger if kept in its place; but when allowed to fly straight from the dung- or refuse-heap to commit suicide in the milk bowl, or alight on the lips of a sleeping infant, or walk over meat and other articles of food on the table, he considered it the most active pathogenic agent during the summer, and the principal cause of summer or epidemic diarrhœa. It is in milk that it proves most dangerous, for it falls into milk bearing on its body and legs such germs as *Bacillus Coli*, *Proteus Vulgaris*, *Bacillus Enteriditis* (Gärtner), *Proteus Zenkeri*, *Streptococci*, *Staphylococci*, and other pathogenic organisms. The bacteria which he had mentioned have actually been isolated from flies taken from a refuse-heap, and examined by Mr. Foulerton at the

Middlesex Hospital. By the aid of flies the Eberth-Gaffky and diphtheria bacilli may easily be conveyed to milk. In milk bacteria find an ideal pabulum and multiply at a astonishing rate, particularly when the atmospheric temperature is high. This had been shown by various observers, and quite recently at the December meeting of this Society it was emphasised by Professor Delépine, in his valuable contribution on "The Bearing of Outbreaks of Food Poisoning upon the Etiology of Summer Diarrhœa." This contribution was of the highest scientific importance; and he thought most would agree that, in view of the number and nature of Professor Delépine's observations, he was justified in his general conclusions as to the chief cause of epidemic diarrhœa being fœcal contamination of milk at the farm or in transit. All sanitarians would cordially endorse the preventive measures he mentioned: indeed, most Medical Officers of Health already strove to secure this cleanliness of dairy premises, cowsheds, milkers, utensils, etc.

Professor Delépine had substantially corroborated what bacteriologists for some years have recognised, viz., that time and temperature were most important factors in the multiplication of bacteria, and, consequently, the degree of infectivity of milk.

Some samples of milk were no doubt contaminated by the use of unboiled polluted water for washing the utensils, as in the Victoria Park, Manchester (1894) epidemic; this was an instance of direct infection of milk by human agency. The point, however, that he wished accepted was that in his belief *flies* are the chief agents concerned in carrying fœcal pollution to milk during the summer months, and are *facile princeps* the chief indirect casual agents in summer diarrhœa, just as mosquitos are the chief indirect causal agents of intermittent or malarial fever.

His experience of epidemic diarrhœa at Southend during 1902 had given him some ground for advancing this hypothesis. During July and August there was no mortality among infants under one year from diarrhœa, as compared with twenty-three deaths from the same cause during the same period of 1901. July and August, 1902, were comparatively cool months, and rain fell on twenty-two out of the sixty-two days (there being thirteen wet days in August). The most remarkable phenomenon of these months was, however, the almost complete absence of *Musca domestica*. In September this insect made its appearance, and, coincidentally, epidemic diarrhœa made

its appearance, and thirteen infantile deaths from this cause were recorded in three weeks. Unfortunately, there was no 4-foot earth thermometer in Southend at the time, but he doubted if the earth temperature reached that point. The advent of the fly was of short duration, and the insect had practically disappeared again by the 25th of September. From this date no further deaths from diarrhœa were recorded in Southend until November, when a solitary death from enteritis appeared in the death returns.

Where refuse and midden-heaps were in the proximity of cowsheds and dairies, they were, he was sure, a source of great danger, even if the water supply was pure and beyond suspicion. He thought they were a danger partly, and to a lesser degree, by the risk of infective dust being blown on to exposed milk during dry weather; and partly, and to a greater degree by, the risk of flies carrying contamination direct from the refuse-heap to the milk, or to the udders of the cow, or the hands of the milker. Subsequent to this method of infection, time and temperature (as shown by Professor Delépine) played an important part in the increase of infective material, not to speak of fresh contamination by flies and dust in the dairy shops and in the homes of consumers.

In addition to the all-important preventive measures which should be carried out at the various cowsheds and dairies, as formulated in Professor Delépine's paper, Dr. Nash would add *the covering over of all standing milk, so as to absolutely prevent the access of flies.* How often one saw a large uncovered bowl of milk on a counter, labelled "pure milk," exposed to the dust, and with perhaps one or two flies floating in it, and several others skirting its edges.

In the house and home it was still essential to insist on the observance of all measures which conduce to cleanliness, and the protection of milk, etc., from contamination. In June, 1902, he published in Southend-on-Sea a pamphlet, containing certain rules and precautions to be observed by mothers of households. Some of these had been insisted upon previously by various sanitarians. The rules were, he thought, so important (though so obvious and simple), that he made no apology for recording them in a paper read before so learned a Society as this. He had just added to these rules certain additional precautions on the importance of excluding flies from milk, etc. The taste of boiled milk was very generally objected to. The method of meeting this objection, as given in Rule V (2), was

very useful and perfectly satisfactory. The milk was heated up to over 200 deg. Fahr. for twenty minutes, but did not itself boil. In connection with this matter, a recent discovery by Dr. Gærtner* was of considerable value and importance. He found that the peculiar taste of boiled milk depends upon contact with the air, and that it could be prevented by the addition of a small piece of paraffin wax to the heated milk. A surface air-tight layer of paraffin forms on the milk, which can be removed afterwards. Paraffin is insoluble in milk and without odour; its action is, therefore, purely mechanical and unobjectionable.

MAJOR FIRTH, R.A.M.C., was afraid that he knew very little about the subject of this paper, and he was sorry not to have been present last month, He had only been able to run over some of the chief points of the paper, and could only grasp some important points of Dr. Newsholme's criticism. It seemed to him that the chief point advanced in the original paper was that epidemic diarrhœa was due to some of the *coli* group, with which group he had had considerable experience; they were some of the most difficult to deal with. He joined issue with Dr. Newsholme as to the agglutinative action. Dr. Delépine stated that the *coli* group might be the incriminating organism, but he thought the case was not proved. His experience, which was limited to the service in India and the tropics, was markedly in contrast with that of the civil population. They had very little diarrhœa, although the sources of the milk supply were ten times worse than in England. His examinations brought him to the conclusion that milk, even if swarming in *coli* organisms, does not produce diarrhœa. He agreed that inflammation of the udder does not produce diarrhœa. Flies were often productive of much danger. He was disposed to think that we were in a state of crass ignorance as regards epidemic diarrhœa. He did not think that it was due to the *coli* group. He thought we must still work to find out to what it is due. It was clear, he thought, that it was an organism concerned with the excreta. There was another point which struck him: that we were inclined to think that all diarrhœa in children was due to bacilli. We could not say that epidemic diarrhœa was due to any particular cause. We must keep on working till we got to the bottom of it.

* Quoted in the *British Food Journal*, December, 1902.

MR. GOADBY thought that only a bacteriologist could appreciate the vast amount of work involved in examining guinea-pigs. We must keep an open mind as to the bacilli of the *coli* group. They were present in a very large number of samples of milk examined. It would be valuable to have information in the paper as to whether milk had been injected directly or not. It was possible that some *coli* were pathogenic and some not. He thought it would add to the interest of the paper to know the methods adopted in the isolation of these organisms. One would expect to find *coli*, being an organism constantly present. His experience was in accordance with Major Firth's. His experiments showed him that agglutinative reaction varied in a very remarkable manner.

DR. REGINALD DUDFIELD regarded Professor Delépine's paper in one sense as the charter of the cowsheds. It was a grand demonstration of the need of cleanliness in cowsheds. Dr. Dudfield had had ptomaine poisoning on two occasions. The first was in the summer of 1894, as a result of eating dried haddock; the London variety is often fit only for the dust-bin. The second time was the result of partaking of pigeon-pie. The first day the pie was eaten with impunity; but, on the second day, he and a relative had some, and the result was ptomaine poisoning. With regard to milk. Two or three years ago, he sent his family to a hamlet near Cheltenham. The people of the farm had their own cows, which were driven in every day to be milked to a cowshed, which he was sure was inches deep in cow-dung, and the walls were covered with the same material. The milk was supplied to the farm and to the chief dairy of Cheltenham. He should think there was good scope there for *coli*. On the Great Western Railway, milk cans are often left standing on the platforms exposed to the sun and dust, and then, perhaps, put into trucks or into the guard's van, where dogs, possibly, accompanied them: he thought it very reprehensible. The milk should go into its own carriages, specially reserved for it. With regard to summer diarrhœa, in London at least, there was a largely increased use of condensed milk: he could not understand why, except that it was cheaper; some of it was by no means satisfactory. With reference to increase of bacilli, it is well to bear in mind Dr. Hope's observations on increase of diarrhœa in heavy rain. Dr. Dudfield made some inquiries a few years ago at Eastbourne. In about 99 per cent. of the deaths the children were fed by bottles.

He had the same experience at Paddington. One of the previous speakers had referred to flies; he had a similar experience. In the North Walk, Paddington Basin, a man came from Bristol, and thought he could "run the show," when we were suffering from a plague of flies. He had methods of his own, which were unsatisfactory, and quickly, under pressure, he vacated the premises: there had been less enteric since. Flies were singularly few last summer. Major Firth referred to the disposal of excreta: London was not troubled much in this direction. Referring to the disposal of house-refuse, one often saw in a low-class street festering dustbins awaiting removal, which occurred once a week, or perhaps less often. The temperature went up to 54 deg. He did not think one could look upon that as cause and effect. If that were so, he should expect high infant mortality to follow.

DR. PAYNE referred to the historical aspect of our knowledge as to summer diarrhœa, and pointed out that Sydenham clearly recognised the disease in his writings in the seventeenth century. Dr. Payne then invited Professor Delépine to reply to the criticisms which had been made upon his paper.

PROFESSOR DELÉPINE: I have to thank the members of the Society for the trouble they have taken to study the evidence I have brought before them, and for the kindly criticism to which they have submitted my views. The nature of the arguments which have been brought forward shows that current ideas regarding epidemic diarrhœa are far from clear; and this is, I think, sufficient to justify even such an imperfect attempt as mine at bringing together facts which seem to have some bearing upon the etiology of epidemic diarrhœa. These facts may possibly prevent the attention of observers to be so completely and persistently given to the soil, and specially to the 4-foot thermometer, and induce some to consider more fully the importance of more superficial and accessible objects.

I wish to make it clear at once that, however convinced I am of the importance of cow's milk as a source of infantile diarrhœa, and of the dangers resulting from the infection of milk before it reaches the consumer, *I do not ignore the existence of other causes of epidemic diarrhœa.* If I can explain the way in which infection is produced in two-thirds or even only half of all cases, I will be quite satisfied.

If by stating my case too strongly, or by being silent upon certain facts, I have misled anybody, I have done so inadvertently, and hope I shall be excused for my shortcomings.

I am very much indebted to Dr. Newsholme for the care he has taken to consider most of the points I have raised, and for many of his suggestive remarks which, coming from such an authority, are very valuable.

There are certain parts of my thesis which he accepts as proved; these may be summed up as follows:—

1. *Food infection* is the chief source of epidemic diarrhœa.

2. *Milk is the food* most liable to act as the breeding-ground of the disease.

3. *The infection is generally due to fæcal pollution*, by which entrance is given to *bacilli of the colon group.*

These are my most important points, and it is a matter of satisfaction to me that they do not require further discussion.

Dr. Newsholme, without entirely disagreeing with me with regard to other points of less importance, believes that I have underrated the influence of certain factors and exaggerated that of others.

1. With regard to *temperature and rainfall*, whilst I contend that high temperature (or temperature *plus* time) is a factor second only in importance to the actual infection of food, Dr. Newsholme, on the other hand, considers that *the incidence of diarrhœa is more influenced by rainfall than by the mean temperature of the air.* This he attributes to the removal of infective material from the ground, and interference with the movements, if not the actual hatching, of flies.

I do not deny the influence of rainfall upon epidemic diarrhœa, and I am quite convinced that rain cleans the ground, allays dust, and affects flies. I think, however, that with regard to flies it would be desirable to know what becomes of them during rainy days, and whether they are hatched rapidly enough to account for the sudden rise in the incidence of diarrhœa which follows when a few wet days are succeeded by a few hot days during the diarrhœal season. Any estimate of the influence of rainfall upon disease based upon yearly averages is misleading. To obtain a true insight into the influence of temperature and rainfall, it is necessary to compare the number of cases occurring *daily or weekly* with the temperature and rainfall of the corresponding days or weeks. It is then found

that diarrhœa does not become *prevalent* till a summer temperature has been reached. When the disease has become prevalent, the number of cases occurring is almost directly proportional with the temperature. It is true that when rain falls for two, three, or more days, one notices that there is a diminution in the number of cases; but there is also a rapid lowering of temperature due to the obstruction of the sun's rays by the clouds, to the coldness of the rain-water, and to evaporation. The influence of rain is so rapid that it cannot be explained by its action on flies or upon the deeper strata of the soil. The only sudden effect which rain might have upon flies would be to drive them from the open to sheltered places, which would not account for a rapid diminution of cases on the supposition that flies are the disseminators of diarrhœa germs. It seems also obvious that such a sudden effect could not be attributed to the interference with the hatching of these insects. On the other hand, it is clear that any cooling of the air, and consequently of the railway and other vans, milk cans and their contents, dairies and farms, interferes rapidly with the multiplication of the bacilli which I have found to be present in infectious milk. These bacilli multiply slowly at the temperature, prevalent in winter and spring, but proliferate with such an extraordinary rapidity at summer temperature, that *in a few hours* a large mass of milk may become entirely infected by a small particle of infectious matter. In my opinion, therefore, the theory which I have proposed is entirely supported by the influence which temperature and rainfall have upon the incidence of epidemic diarrhœa, and I am still of opinion that of *the accessory factors favouring infection temperature is the most important.* And yet I agree with Dr. Newsholme regarding the danger of attaching too much importance to Dr. Ballard's critical temperature. The 4-foot thermometer is not sensitive enough to sudden changes of superficial temperature to be used for any other purpose than that of indicating great seasonal variations. As a means of averaging temperature by giving an equalised curve it is very useful. Its indications allow one to ascertain the relation between temperature and the seasonal mortality from diarrhœa. The 4-foot thermometer does not, however, give any indication with regard to the sudden exacerbations of incidence due to the sudden rises in the temperature of the air. Ballard's critical temperature is, therefore, misleading only if one attaches to it a meaning which it has not; specially if this supposed

meaning direct attention too much to what is taking place in the soil itself.

2. Dr. Newsholme attaches more importance than I do to the absence of any definite *relation between the incidence of diarrhœa and various milk supplies.*

With regard to breast-fed children, it is well shown by all statistics that these children are less liable to diarrhœa than children fed on cow's milk; this is obviously in agreement with my views, and I believe that if the children fed by their mother *never received anything else than their mother's milk, the number of cases of diarrhœa among breast-fed children would be still smaller than it is.*

With regard to the fact that it is usually difficult to connect outbreaks of epidemic diarrhœa with the milk supplied by certain dairies, this does not, as Dr. Newsholme is inclined to believe, affect the question at all. I have adduced evidence showing that, under certain conditions of temperature and keeping, *nearly all the samples* of milk reaching a town had infectious properties, and that the same kind of milk, when kept cool, was seldom infectious at all; it therefore appears to me that supply areas must have but little influence in themselves upon the distribution of epidemic diarrhœa. What is important to know is the time which has elapsed between the milking of the cow and the drinking of the milk, and also the temperature of the milk during that period. In this respect, summer diarrhœa differs from limited outbreaks of food poisoning, or such a disease as typhoid fever; in the latter cases an *exceptional opportunity for infection must have occurred,* and it is obvious that such a special opportunity cannot occur at many dairies at the same time. We would, therefore, expect an outbreak of milk typhoid or of typical food-poisoning to be connected with the supply from one farm or dairy only. On the other hand, if my theory is correct, *one would not expect epidemic diarrhœa to be connected with any special dairy,* unless that dairy was conducted with so little care as to attract at once the attention of sanitary authorities by causing also well-defined outbreaks of food poisoning.

As to the influence of the *mode of carriage,* this is worthy of serious consideration; but, under the present conditions, I do not think that one can attach any importance to the mode of transport, unless one takes also account of the time taken in transit and of precautions taken against heat. Milk sent to a town by road in a farm cart may take as much time, and be as much heated and jolted, as milk

sent by rail from a much greater distance. Statistical returns relating to road-borne milk should, therefore, include many particulars before they can be correctly interpreted.

I do not think that *the use of condensed milk* excludes the possibility of farm or factory pollution. *Many kinds of condensed milk are rich in dirt and bacteria* before they reach the consumer. These bacteria do not multiply in the thick syrupy fluid in which they may be said to be in a *state of suspended animation;* but the case is not the same after the milk has been diluted. The bacteria present in condensed milk are derived from the farm, the milk vessels, and probably the factory also; until we know more about the properties of these bacteria, it seems to me impossible to argue anything from the effects of feeding on condensed milk.

Although I have defended my position by trying to prove that the facts upon which Dr. Newsholme based his arguments in favour of domestic infection are capable of interpretations which favour my own views, I wish once more to state that I do not deny the occurrence of home infection, but that I think its importance secondary to that of infection at the farm and through dirty, infected milk vessels.

Dr. Newsholme suggests the use of the *serum reaction* for the purpose of completing my chain of evidence. I would entirely agree with him if I had found any satisfactory method to make this reaction practically useful in the investigation of summer diarrhœa; unfortunately, I have not, as I have already stated in my communication, a fact which Dr. Newsholme has probably overlooked.

When I introduced Widal's reaction during the month of July, 1896, into this country, I naturally thought at once that it might become very useful for the purpose of investigating the etiology of epidemic diarrhœa. I immunised small animals against bacilli of the bacillus *enteritidis* and bacillus *coli* types. With the assistance of Dr. Brindley, who was then working in my department, I tested a number of blood samples. We obtained clumping reactions with the blood of immunised animals, and of some patients. Unfortunately, the reaction appeared at various intervals after infection, and in some cases disappeared very rapidly, and was not to be compared in sharpness with the typhoid reaction.

Even in connection with such a clear outbreak of food poisoning as the one I have lately investigated in Derby,

Dr. Sellers, Dr. Carver, and myself have found that only a certain number of individuals gave a clear reaction with bacilli obtained from the food which had caused their illness, or from the blood of other patients who had been infected with the same food. In cases where a good reaction had been obtained, the reaction was observable only during short periods. In addition to this, there are several difficulties which I need not detail here. I think, therefore, that until better results can be obtained by the serum test, it will still be necessary to adopt (as I have done) the laborious process of isolating the bacilli, testing their cultural properties and their action upon animals, in order to obtain accurate data.

Dr. Meredith Richards thinks that if temperature is such an important factor as I believe, cow's milk should become infectious whenever there are a few days of hot weather; he finds, however, that a few hot days frequently occur without there being any epidemic diarrhœa. I think that sporadic cases of epidemic diarrhœa are frequently overlooked, and that attention is paid to *epidemic* diarrhœa only when the disease assumes epidemic proportions from the continuance of hot weather.

When I say that the most infectious samples of milk had an *alkaline or amphoterous reaction,* I do not mean to say that the supply of sour or clotted milk, or of milk likely to become sour in a few hours, is not objectionable—all I wish to advance is, that the reaction of cows' milk is no sure index of its wholesomeness or infectiousness.

I recommend cooling of the milk down to 4 deg. Cent., because this stops almost entirely the multiplication of the objectionable bacteria. Cooling down to 10 deg. Cent. is safe enough in theory, for multiplication takes place comparatively slowly at that temperature; but it is not safe, for practical purposes, to fix the temperature at a point so near that at which danger begins to occur; for the dangerous bacteria begin to multiply rapidly at 13 deg. Cent.

In answer to Mr. Goadby, I may say that, *except in cases where milk was the obvious cause* of limited epidemics of food poisoning, I have not attempted to separate the bacilli of the colon group direct from the milk. Such a procedure would have been lengthy and unpractical in the case of ordinary milk, where one finds always a large number of more or less innocent members of the colon group, as well as many allied bacilli. I have chiefly studied the bacilli isolated from the blood and tissues of guinea pigs that had

died from what appeared to be a septicæmia, resulting from inoculation with milk. I have, by this method, excluded from consideration those bacilli which were not virulent, or had only a slight virulence, and confined my attention to those bacteria which were virulent, as proved by the results of inoculation.

In conclusion, I beg to thank the President and members of the Society for their patient hearing and kindly treatment.

SOME OF THE DANGERS OF BORACIC ACID AND FORMALDEHYDE AS FOOD PRESERVATIVES.

BY S. DELÉPINE.

THE use of boracic acid and formaldehyde, even in small proportions, for the purpose of preserving food, and more especially milk, is objectionable, and a source of danger to the health of the public.

My opinion is based on the following facts :—

I.—In *large doses these substances have been proved to act as poisons.*

To this it may be said that the amount of preservatives added to food is always small when compared to poisonous doses; but when it is remembered that several people, *e.g.*, the original vendor, the retailer, the cook, may all in succession add the same preservative to the same substance, it will be evident that comparatively large doses may ultimately find their way into an article of food. The only safeguard that can be offered to the consumer is the entire *prohibition of the use of preservatives;* the selling of which as such should also be prohibited, *i.e.*, the advertisement for sale of food preservatives.

II.—Even when preservatives are used in doses just sufficient to effect temporarily their purpose, *i.e.*, 35 grains of boracic acid to the gallon of milk, and about 1 grain of formaldehyde per gallon (1 in 100,000 to 1 in 50,000), it cannot be said that the addition of these chemicals is without danger.

A. There have been cases on record in which illness has been traced to the use of boracic acid. It is well known that some people, especially *infants and invalids*, are *much more liable to the action of such drugs* than average individuals. The fact, therefore, that many people can take large doses of boracic acid and formaldehyde without apparent bad effect is no proof that everybody should be exposed *without knowing it* to the action of these drugs.

B. Experiments by various observers have yielded various results, but there are several series of experiments on record showing :—

1. That the health of young animals is seriously affected by the consumption of milk containing boracic acid or formaldehyde.
2. That the digestive processes are retarded by the use of these drugs.
3. That proteid substances are rendered less soluble.

C. Even if one accepted as reasonable the doubts which have been thrown upon such experiments, it is obvious that the consumer must be given the opportunity of determining whether he cares to be submitted, or to have his children submitted, to experiments; and, therefore, *milk containing boracic acid and formaldehyde should be sold as such, the buyer being warned of the presence of the drugs; it should not be sold as fresh milk.*

III.—*The use of boracic acid and formaldehyde* is also to be objected to, because it allows the vendor to sell to the consumer *a stale and often dirty article instead of the fresh article paid for.*

When cows are milked under proper conditions of cleanliness, when the milk is stored in clean cans, is kept cool, and is distributed as rapidly as it should be, the milk keeps quite long enough for all practical purposes. Good clean milk, stored in a cool place, keeps well for forty-eight hours at least. In a refrigerator, milk can be kept good almost indefinitely.

A. *The addition of boracic acid retards the lactic acid fermentation, and the clotting, which to the consumer are the usual signs whereby he knows whether milk has been kept too long or is bad in quality.*

I have tested carefully the amount of this retardation and its effect on milk, with the following results:—

At the ordinary temperature of the room (15 deg. Cent.) the addition of boracic acid to fresh milk had the following effects:—

Quantity of Boracic Acid (and Borax) added per Gallon.	First appearance of distinct Acid reaction after.	First appearance of Clotting after.	Bacteria became innumerable after.
None	2 days (slight)	3 days	1 day
70 grains ...	3 days (slight)	4 days	1 day
140 grains ...	3 days (very slight)	5 to 6 days	2 days.
700 grains ...	5 to 6 days	7 to 11 days	11 days
1,400 grains ...	5 to 7 days	7 to 11 days	Still few after 15 days

Temperature 15 deg. to 16 deg. Cent.

B. By keeping milk at the temperature of a cool cellar (about 11 deg. to 12 deg. Cent., which is about the temperature of the water in the Manchester mains), *fresh, clean, untreated milk could be kept for four days at least without clotting.*

From this it is obvious that the addition of boracic acid to milk has for its chief object the retardation of coagulation, and that a much larger amount than is usually acknowledged is needed to obtain a practical result in this direction. It is also clear that the same result can be obtained by simple care and cleanliness.

IV.—*The prevention of clotting by the use of preservatives may be a source of very serious danger, by favouring the consumption of milk containing bacteria capable of producing typhoid fever and food poisoning.*

Whilst the addition of borax and boracic acid checks to a certain extent the growth of bacteria producing acid fermentation, it does not check appreciably the growth of bacteria, such as those of typhoid fever and those producing certain forms of inflammation of the bowel, which are often fatal. Then, bacteria do not grow well in acid milk, whilst they grow rapidly in alkaline milk.

This I have tested by direct experiments.

Effects of the addition of Boracic Acid upon the growth of the Bacillus of Typhoid Fever, and of the Bacillus isolated from Provisions: Pork Pies, Milk, and Cheese (Bacillus Enteritidis).

Quantity of Boracic Acid.	Growth of Bacillus Typhosus.	Evidence of Growth of Bacillus Enteritidis.
35 grains per gallon ...	Fairly abundant after 1 day	Abundant in ½ day
70 grains per gallon ...	Fairly abundant after 2 days	Abundant in 1 day
140 grains per gallon ...	Fairly abundant after 2 days	Abundant in 2 days

Effects of the addition of formaldehyde to fluid media upon the growth of the bacillus of typhoid fever, and of the bacillus coli communis in these media. Upon this point I have made some experiments also, and have found that it was necessary to add more than 7 grains of formaldehyde per gallon to check certainly the growth of the bacillus of typhoid fever, and that a longer dose was needed to arrest the growth of the *bacillus coli communis* in fluid media.

SUMMARY.

V.—From all this I conclude that the addition of preservatives is a serious source of danger to the public, and should be prevented.

1. It is unnecessary.

2. It favours the sale of stale or dirty articles, by hiding defects which would otherwise be apparent.

3. It may be directly detrimental to the health of certain individuals.

4. It does not check the growth of certain infective bacteria, which, owing to the milk not becoming sour, have time to multiply to a dangerous extent before the milk is consumed. This danger would be less if preservatives were not used, for the milk would become obviously bad at a much earlier period.

THE RELATION OF THE PANAMA CANAL

TO THE

INTRODUCTION OF YELLOW FEVER INTO ASIA.

By DR. PATRICK MANSON, C.M.G., F.R.S., Medical Adviser to H.M. Colonial Office; Ex-President of the Epidemiological Society.

(*Read: February 25th*, 1903.)

THE influence of trade and travel routes on the diffusion of disease is a fascinating and important branch of epidemiology. But it is an extensive and intricate one, and I have no intention of attempting its discussion; at all events, in its wider bearings. My object on the present occasion is much more restricted: it is to elicit an expression of opinion on the part of the members of this Society on two specific points connected with the wider subject. These are: 1st, The risk of the introduction of Yellow Fever into Asia that will attend the making and working of the projected Central American Inter-Oceanic Canal; 2nd, The measures that should be taken to lessen the risk of such a calamity.

It seems to me, that the present is a suitable time to bring forward this matter; for it is a matter which, in my humble opinion, is not only one of importance, but also one of urgency. Providentially, recent discovery has given it a hopeful and practical aspect which, but a few years ago, it did not possess. Whereas, formerly, we knew little or nothing of the way in which Yellow Fever was conveyed, we now have a body of exact knowledge to guide us, and to work on. There can be no question about the urgency of the matter. The United States of America are pledged to carry through a Panama Canal. Their national pride is at stake. Their economic as well as their Naval interests would be so manifestly favoured by the success of this great undertaking that, apart from considerations of national ambition, we may be certain that every effort will be made to carry the scheme to a successful issue. Already, the purchase of the old workings has been agreed on. In a year or two, perhaps in a month or two, active opera-

tions will commence. Ship-load after ship-load of labour in the shape of Chinese, Japanese, or Indian coolies, will be landed at Panama, and steamers to supply the peculiar wants of the coolies will be plying backwards and forwards between Central America and the ports of Asia. So soon as the works begin, the risk that some of these steamers may convey Yellow Fever to Asia will begin. To put off sanitary measures, therefore, until the Canal is completed is, in my opinion, to incur a terrible risk. It might be too late then. The calamity I contemplate might have already fallen on the Asiatic world.

I firmly believe that, if direct and rapid marine communication between the present endemic area of Yellow Fever and Asia be permitted, and, if at the same time, efficient precautions against the conveyance of the Yellow Fever germ be not instituted, in a very short time there will be Yellow Fever in Asia. I also firmly believe that a properly devised and administered sanitary organisation could prevent such a catastrophe. We have the necessary knowledge; we need only apply it. It will be a standing reproach to the sanitarians and Governments of this generation if, knowing the risk, and knowing how to counteract it, Yellow Fever be allowed to get a footing in Asia; if, having the power to prevent this, they did not have the will or the wisdom to use this power.

That the introduction of the germ of Yellow Fever into Asia would be a disaster of the first magnitude cannot be doubted. We know what Yellow Fever did for Spain, for the United States, for Brazil, and for the Argentine. We know what it has done for the West Indies. We know that on more than one occasion it has wiped out French and English armies. We know the heavy toll it has levied on European, Chinese, and Indian life in Havana, in New Orleans, in Rio de Janeiro, and in many other cities in that part of the world. We know how the fear of it has taxed and dislocated commerce. It is hardly necessary to mention these things to this Society. If Yellow Fever has done so much mischief in the relatively restricted and not very densely populated endemic and epidemic area in America and Europe, how much more serious would its ravages be were it introduced into Asia and the island dependencies. There the germ would fall on an absolutely non-immune community, with numberless cities and densely populated areas, linked together by an active but unregulated commerce. It would spread from trade centre to trade centre. It would enjoy opportunities even more favourable than it

has ever encountered in America or South Europe; for the cities of Asia are larger, older, more densely populated, more insanitary; with inhabitants wedded to old customs and prejudiced against sanitary methods; passively, and sometimes actively, hostile to all sanitary and municipal reform. The dislocation of trade would be ruinous. Imagine what would happen if Yellow Fever broke out in distributing centres such as Zanzibar, Aden, Bombay, Calcutta, Colombo, Singapore, Batavia, Bankok, Saigon, Hong Kong, Shanghai, or Yokohama. Trade, in its wider sense, would stop. Our merchants, manufacturers, and shipowners, both at home and in Asia, would be ruined. And, if after a time, the initial epidemic outburst died down, there would still remain here and there throughout the tropical portion of the Eastern Hemisphere endemic centres of the disease—Oriental Rios and Havanas. These would be permanent threats to the rest of the Continent, necessitating permanent, and very irksome, and very expensive quarantine and isolation measures. To cholera, dysentery, and malaria, which already nearly everywhere threaten European life in the East, another and perhaps worse danger would be added.

I think in this matter the Epidemiological Society of Great Britain has a distinct duty, and, I would add, a very great opportunity. Just as this Society took a leading part in the fight against small-pox in Britain, so I conceive it might follow a similar course as regards Yellow Fever in Britain's Asiatic dependencies. It can influence public opinion. It can advise and influence the Government.

To proceed with the points I wish to bring forward for discussion:—

1. The risk of the introduction of Yellow Fever into Asia by the making and working of the Panama Canal.

I can imagine that some will set me down as an alarmist and busybody. I can imagine that some will say that, as Asia has enjoyed an absolute immunity from Yellow Fever during all these ages, this immunity will continue for our day at least, and therefore that there is no occasion to bother about it. To such I would answer that before Columbus's time, some Aztec or Inca, had he known about Europe and its diseases, might have said the same about small-pox, cholera, or plague. And so long as their continent was practically isolated from the Old World, as regards the Old World diseases these New World epidemiologists would have been right enough. But so soon as active communication with the Old World was established,

any *laissez aller* policy the Inca-Aztec sanitarian might have founded on this way of thinking would have been wrong. The Panama Canal will do for Asia, as regards Yellow Fever, exactly the same thing that Columbus did for America as regards European and Asiatic epidemic disease; this it will do if we remain Inca-Aztec sanitarians; if we do not recognise the danger and take measures accordingly.

Hitherto there has been no direct communication between the endemic haunts of Yellow Fever and Asia. It is quite true that there has existed for some decades an active communication between Western America and Asia; but this communication has been exclusively between points far to the north or far to the south of the Yellow Fever region, and therefore, in respect to this disease, free from risk. But with the opening of the Canal there will come a change; a huge and increasing commerce will pass through the Yellow Fever region direct to Asia. Hitherto the course of ships has been well to the north or well to the south of the Tropic belt, and generally in cold or temperate zones, in temperatures inimical to Yellow Fever and the distributor of Yellow Fever. But the new route—the direct route from Panama to Japan, China, Manila, the Straits Settlements, Java and Australia—will lie through the Tropics, and, for the most part, in atmospheric conditions favourable to Yellow Fever and its mosquito distributor. Furthermore, the route, instead of being something like fifty days, including transhipments, from Panama to Asia, will be reduced to three or four weeks and include no transhipments, and the coaling ports that will be established in the tropical parts of the Pacific will serve as convenient jumping-off places for the gradual advance of the germ.

That Yellow Fever can be successfully exported, so to speak, from the endemic area, is proved by the various epidemics that have ravaged the South of Europe. These imported epidemics prove that the germ can support a sea voyage, and that other places besides Tropical and Sub-tropical America supply the conditions necessary for its successful propagation. If these conditions are to be found in Europe, why not in Asia? Doubtless they are present in Asia; in fact, we know they are. The only conceivable reason why Yellow Fever has not appeared hitherto in Asia is the circumstance that the germ has never been introduced; and the reason why the germ has not been introduced is that hitherto there has been no direct, rapid, and all-tropic communication between Asia and Central

America. Epidemiologically, the opening of the Panama Canal will inaugurate a new area. The establishment of a great trade and travel route must always be a new factor in the diffusion of disease. It brings with it not only interchange of merchandise, but interchange of germs.

I take it that all are now agreed that the story of the Yellow Fever germ, as disclosed by the labours of Reed, Carroll, Lazear, Agramonte, and Guiteras is correct. We believe now—nay, we know now—that the germ is present in the blood of the patient in a communicable form only during the first three days of the fever; that it is removed from the human body by the *stegomyia* mosquito; that it undergoes a necessary metamorphosis in the body of this insect: a metamorphosis that takes some twelve to fourteen days to complete; that it is transferred to the bodies of non-immunes by the bite of the mosquito; that clinical evidence of the inoculated germ's presence is manifested in from three to five days after inoculation; that an infected mosquito retains its capacity to convey the germ for at least fifty-seven days from the time it bit a Yellow Fever patient. We know, further, that *fomites* have nothing whatever to do with the spread of Yellow Fever, and that our ideas of a few years ago were in this respect absolutely erroneous. There is no well-established fact that can be regarded as a reason for thinking that there is any other way by which, under natural conditions, the germ of Yellow Fever is conveyed than by the mosquito. We have no reason to suppose that the *stegomyia* mosquitoes of Asia, including *fasciata*, which abound there, would be inefficient as an intermediary of the germ. The European species has shown itself to be efficient. Why not the Asiatic?

If we apply this new-found knowledge, we will not only receive the explanation of the heretofore absence of Yellow Fever in Asia, but we will also be able to forecast the effect that changed conditions of communication, brought about by the Panama Canal, will have, or may have, on the future of Asia as regards this disease.

A steamer from Cuba, Rio de Janeiro, or other American port where Yellow Fever is raging, ships among her passengers a *stegomyia* mosquito that has recently made a meal off a Yellow Fever patient. The mosquito conceals herself, as is the habit of her kind, in some dark and quiet nook of the ship. From time to time, during the voyage, she feeds on fruit, sugar, rat, dog, fowl, or such

live-stock as chances to be on board. The ship passes through the Canal, and, in due course, arrives at Manila or Hongkong. Cargo is discharged, cabins, holds, store-rooms, live-stock pens are cleaned out, and the mosquito, being disturbed, takes flight, and reaches the shore. Presently, she has a meal off the blood of some native. With her saliva she injects into him some Yellow Fever germs. Three days later, the native falls sick. His disease is not recognised, and he is not isolated. Local *stegomyiae* feed on him, and then, a fortnight later, on other natives. The fat is now in the fire, and Yellow Fever begins in Asia. The physicians, not having seen the disease before, are some time in apprehending its nature. It is too late then. Hundreds have died or are ill. The mortality is great. Many of the inhabitants flee from the plague-stricken city. Some of them carry the germ with them. So the disease is spread from point, to point, and the epidemic rapidly attains unmanageable proportions. By degrees, the whole of Tropical Asia becomes involved, and we are in the presence of one of the worst and most deadly epidemics the world has known.

Or, there is Yellow Fever somewhere along the Panama Canal. A ship is passing through. An infected *stegomyia* from the shore comes on board. She feeds on one of the crew. Next day, the ship clears from Panama. Two days later, the bitten sailor has Yellow Fever. There are several, or perhaps many, *stegomyiae* in the ship. They feed on the patient. Fourteen days later, some of them bite another, or other, sailors. Three days later, these other sailors have Yellow Fever. There are now many infected *stegomyiae* on the ship. By this time she is near to Asia. The Captain, at his wits' end, puts into the first port he comes to for medical assistance. There is no quarantine or sanitary organisation at the port, and free communication with the shore is permitted. The infected *stegomyiae* fly ashore, and three days later an epidemic of Yellow Fever is in full swing.

But there are other ways by which the infection could be brought to Asia. I can imagine some scientist, more enthusiastic than prudent, deliberately carrying with him, for purposes of study or experiment, infected mosquitoes from America to Asia. Should one of these insects escape, it might very well start an epidemic, We know what has been done in this way with the plague bacillus, and what a narrow escape certain cities—Vienna, for example—had not so long ago.

I can even imagine some malignant Nero-like monster intentionally introducing an infected insect, and gloating over the result. Or I can imagine some wretch, with a perverted craving for fame, like that Erostratus who set fire to the Temple at Ephesus, letting loose in this way a bigger scourge to Asia than ever fire or sword have proved. But apart from risks of this sort, the repeated day-by-day recurrence of opportunities for infected *stegomyiae* being transported to Asia from America, must, if given uninterrupted play, sooner or later bring Yellow Fever to Asia.

It is not as if the Yellow Fever mosquito were a delicate insect, or one peculiar to America. *Stegomyia fasciata* is the most widely distributed and one of the most common of the tropical *Culicidae.* That is so probably because it is the best traveller of its kind, and, therefore, the best suited in this respect for the transport of the Yellow Fever germ. Its range is from Spain and Japan in the north to Natal and Australia in the south. The vitality of its eggs is not destroyed by prolonged (months) desiccation. They will hatch out in the dark, and its larvæ will live in the dark and in foul water. The insect itself is very adaptable in the matter of food, and can live at least two months on water alone (Theobald), and altogether, except as regards low temperatures, is a hardy insect.

I think I have said enough to show that my fears about Yellow Fever are not imaginary, and that it could very easily be carried to Asia, and that once introduced it could flourish there.

I pass now to the second point I propose for discussion, namely, the measures that should be taken to avert the introduction of Yellow Fever into Asia, *viâ* the Panama Canal.

It is manifest that quarantine directed against Yellow Fever *fomites* is useless and unnecessary. Reed and his colleagues proved that the *fomites* of Yellow Fever patients were as harmless as the *fomites* of malaria fever patients. They may be disregarded, therefore; the things to be guarded against are the Yellow Fever patient himself, and the Yellow Fever-infected mosquito.

Now, whatever may be the case as regards North and South America and the West India Islands, where the inter-port distances are relatively short—seldom in these days of steam exceeding, as regards duration of voyage, the combined incubation and infective periods of Yellow Fever, say ten days—it is quite different in this respect with the Trans-Pacific Panama-Asia voyage. This latter is a matter

of several weeks—a month or more. Therefore, quarantine measures directed against the danger from patients infected in America, however necessary they may be for the protection of American ports are quite unnecessary as regards Asiatic ports.

A man might be bitten by an infected *stegomyia* in Rio de Janeiro, go on board a fast steamer the same day, and reach New Orleans before he had ceased to be infective. Or a man might be bitten by an infected *stegomyia* in Havana, and land in New Orleans before he had shown any clinical evidence of a successful inoculation. But such a thing would be impossible in a case of Yellow Fever infection contracted in America and subsequently landed in Asia; the infectivity of such an individual would have disappeared weeks before he landed.

There is no danger, therefore, in shipping Yellow Fever at Panama, and no necessity for quarantine measures directed against Yellow Fever patients shipped for Asia, *on one condition.* This condition, however, is all-important. There must be no mosquito on board the ship in which the Yellow Fever patient travels; no mosquito to keep up and transmit infection, whether to fellow passengers or to the inhabitants of the Asiatic port of destination. Therefore, in dealing with the problem of the exclusion of Yellow Fever from Asia, the Yellow Fever patient may be ignored, provided the mosquito danger be adequately guarded against. And this insect must be regarded as the *crux* of the sanitary situation, whether there be Yellow Fever on board the ship she travels by or not. For, independently of Yellow Fever patients shipped in America, the mosquito may come on board already infected, either at the ports of departure or of call, or during the transit of the Canal. Such a mosquito might very well set up fever on board after the ship had cleared from Panama, at any time during the voyage to Asia, or after the arrival at the Asiatic terminus.

My idea, therefore, in attempting the protection of Asia from Yellow Fever is to disregard the Yellow Fever patient altogether, to have no quarantine, but to stringently enforce destruction of all mosquitoes, their eggs, and their larvæ in all ships, before these ships are allowed to clear from the Pacific end of the Canal, or from other ports on the Pacific side of the American Yellow Fever district. If this be effectually done, Yellow Fever will never appear in Asia.

To carry out this important sanitary measure an organisation and funds are necessary. To ensure its

efficiency, the organisation should be altogether independent of the local Canal functionaries; it must be liberally subsidised; its official head must be responsible to the Secretary of State in Washington, or other central authority, and his position should be such as to secure him freedom of action and freedom from interference by the local authority.

The funds might be provided by a special toll on ships using the Canal, or by a *pro rata* contribution from the Asiatic governments interested.

The chief of the sanitary staff must, of course, be an American citizen—such a man as Gorgas—possessed of ability, experience, energy, independence of character, and, above all, imbued with a thorough belief in the efficacy of anti-mosquito measures. The details of the staff might be selected from those nationalities whose ships are most likely to use the Canal.

That is, in rough outline, the scheme I have in my mind. The particular methods that would be employed to clear ships of mosquitoes can be easily devised after a little experience of the local conditions. I would suggest that every ship on arrival at the eastern end of the Canal be boarded by at least two sanitary officers, who, during the passage of the waterway, would clear her of mosquitoes. Before she puts to sea again at Panama, one or more inspecting officers should board her, and assure themselves that the work has been effectually carried out.

As a supplementary measure, I would suggest that the banks of the Canal, for at least half a mile back, and on both sides, should be cleared of jungle and mosquito pools.

The American people have always—and more especially of late years—shown themselves alive to the value of scientific discovery. They have been prompt, indeed usually the first, to apply it in practice. I have, therefore, very little doubt that the American Government will do all in its power, by anti-mosquito means, to render the making and working of the Canal as little dangerous to those engaged as possible. Not to mention humanitarian considerations, it is the interest of America to do so. But, apart from trade, America has little direct interest in Asia, and we have no right to expect that she will regard the health of the peoples of that Continent as specially in her keeping. It is otherwise with Great Britain. Our stake in Asia is an enormous one; our duty to our fellow subjects there is a very manifest one, and the duty of our rulers to guard our

military and commercial interests there is also a very manifest one. I would suggest, therefore, that Great Britain approach the United States of America, and that together they concert measures for the safeguarding of Asiatic countries against the introduction of Yellow Fever by the Panama Canal. Doubtless Japan, China, France, Siam, Holland, Germany and Australia, all of them directly interested in this matter, would willingly assist in any wise measures which might be devised to protect them or their Colonies from this worst of epidemic diseases.

The world owes an incalculable debt to the American Commission who discovered the bearing of the mosquito on the spread of Yellow Fever. This Commission has given the right cue to the prevention of this disease. Every one of the members of that Commission risked their lives in carrying out the necessary researches and experiments. One of them—Dr. Lazear—succumbed, a martyr, at his post. It must be a matter of profound regret to every one interested in the epidemiology of Yellow Fever that yet another—the leading spirit, as I understand—of this band of courageous workers should have died so soon after the completion of his notable labours, and before he had reaped the full measure of reward to which he was so justly entitled. Dr. Walter Reed did a great and beneficent work. We in England thoroughly appreciate this, and heartily sympathise with America in the loss she and the world has sustained by his premature death. The best tribute we can pay to his memory is at once to apply his discovery. Let us hope that the good he has done will not be interred with his bones, and that his countrymen and the rest of us will take care to push forward the great and beneficent measures his brilliant labours so clearly indicate.

I believe that within the next few years, and in great measure in consequence of the object-lesson this Panama business is about to afford, we will witness a profound revolution in tropical sanitation. The discovery of the part played by the mosquito in the diffusion of three of the most important tropical diseases, although admitted practically by all in an academic sort of fashion, has, so far, with one notable exception, not been applied in a practical and adequate way. Either from ignorance, or indolence, or indifference, or the dread of expense, or from hide-bound conservatism, the discovery has been nearly a barren one. There are not a few who oppose the new idea simply because it is new: because it has the impertinence to afford the explanation of what they had grown up to regard as a mystery

and inexplicable like other visitations of Providence, or because it has upset preconceived and erroneous ideas. There is another order of mind that seems to think that because the entire field of the relation of the mosquito to *filariasis*, malaria and Yellow Fever has not been exhaustively explored and worked out, practical action founded on the ascertained relationship should be delayed. Such a line of thought in any other department of human affairs would be deemed ridiculous. When our neighbour's house is on fire and our own is threatened, we do not wait to study the chemistry of combustion before we send for the fire-engine.

In urging elsewhere the line of action against Yellow Fever which I have advocated to-night, I have been met with the objection that, although these discoveries about the mosquito may be interesting, perhaps promising, they are still far too crude and incomplete for the country to put its money on. Let me call your attention to the Table on the following page, lately published by Gorgas.

Up to 1900, and yearly for one hundred and fifty years, Yellow Fever has claimed in Havana its annual toll of hundreds or thousands of victims; it has not missed one year. In 1900 Reed and his colleagues proved that the mosquito carried the germ. Gorgas promptly applied the discovery. These columns of cyphers are ample testimony, not only to his energy and ability, but to his wisdom. He did not wait till the whole story of the mosquito was worked out. He did not wait till someone should have proved, years hence, perhaps, a negative—that there is no other way by which infection is conveyed. He did not wait even till the germ itself was discovered; it is still unknown. It sufficed for him that Yellow Fever is conveyed by the mosquito. He acted on the ascertained fact, and thereby undoubtedly saved hundreds, if not thousands, of lives, and much treasure.

This is an object-lesson that must not—that cannot—be ignored. The struggle against malaria (which I feel sure the Americans will enter on with keenness and energy, and, I doubt not, with success), at the making of the Panama Canal will be another object-lesson in the application in a business way of scientific discovery, But we may not wait for this second object-lesson. The danger to Asia from Yellow Fever is imminent. Action must be taken at once. Why delay what Gorgas has proved, in so practical a manner, is not only easy of accomplishment, but all-sufficient?

Years.	1890 and 1891.	1891 and 1892.	1892 and 1893.	1903 and 1904.	1894 and 1895.	1895 and 1896.	1896 and 1897.	1897 and 1898.	1898 and 1899.	1899 and 1900.	1900 and 1901.	Average.	1901 and 1902.	1902.
April ...	13	5	8	8	4	6	14	71	1	2	0	12.00	0	0
May ...	23	7	7	23	16	10	27	88	4	0	2	18.81	0	0
June ...	38	41	13	69	31	16	46	174	3	1	8	40.00	0	0
July ...	67	66	27	118	77	88	116	168	16	2	30	70.45	1	0
August ...	60	66	67	100	73	120	262	102	16	13	49	84.36	2	0
September	33	65	70	68	76	135	166	56	34	18	52	70.27	2	0
October ...	22	48	54	46	40	102	240	42	26	25	74	66.27	0	0
November	15	24	52	28	23	35	244	26	13	18	54	48.36	0	0
December	9	17	33	11	29	20	147	8	13	22	20	29.90	0	0
January ...	10	15	15	7	15	10	69	7	1	8	7	14.90	0	0
February .	3	10	6	4	4	7	24	1	0	9	5	6.63	0	0
March ...	4	1	4	2	2	3	30	2	1	4	1	4.90	0	0
—	307	365	356	484	390	552	1385	745	128	122	302	466.90	5	0

To have a thin red band across the Isthmus of Panama, with the Canal in the middle of it, would gratify my patriotism. I am sorry the Panama Canal is not to be a British work. But I must confess that my regret is tempered by the knowledge that it is an American one: for I feel more confidence in American energy as regards

sanitary matters than in our own. We are too slow, too timid, too niggardly, too much afraid of failure, too unbusinesslike. I like the prompt and autocratic way in which the great democracy deals with these things. America, ever since her Colonial expansion began, only a few years ago, whether in Honolulu, or in Cuba, or in Manila, has shown an intelligence and practical appreciation of the benefits of medical and sanitary science, and an energy in these things to which we in England are not always accustomed. I cannot but feel, therefore, that in this matter of the Panama Canal the public interests are in the best hands. Still, this does not free us from our obligations to India, to Australia, and the East generally; and I trust that this Society will do something, by expressing its views freely, to strengthen the hands of Government; or, if necessary, to stimulate our Government to back up America in any measures that may be proposed or instituted with a view to keeping Yellow Fever out of Asia.

Discussion on Dr. Patrick Manson's Paper.

Dr. Strong (Director of the Biological Laboratory in Manila) in a communication on "The Panama Canal and its Relation to the Introduction of Yellow Fever into our Eastern American Possessions," said that the question obviously, as Dr. Manson has fully explained this evening, is one of colossal importance to the Far East. In our American possessions, the Hawaian Islands, Guam, and the Philippines, we shall have to be continually on our guard against the importation of cases of Yellow Fever or of infected *stegomyia fasciata*. During the past year Yellow Fever has been frequently present in Panama, the proposed western terminus of the Canal. Thus, from April 22nd to May 12th, 1902, there were 8 cases and 6 deaths reported; while from July 1st, 1902, until the middle of January of this year, according to the Health Reports of the United States Marine Hospital Service, there have been 133 cases and 23 deaths of Yellow Fever in this city.

As has long been recognised, the disease often follows the lines of commercial maritime travel. Of the frequent introduction of the malady into hitherto uninfected regions by vessels, it is well known there are many instances. Good examples of the conveyance of the disease by ships over short distances may be seen in the infection of the ports

of the Southern United States and Mexico from the West Indies. Whether the Indian Islands were primarily infected from West Africa, or *vice versâ*, must still remain uncertain. The first accredited record of Yellow Fever in Spain is that of 1730, when the disease was imported into Cadiz by a ship from Central America. The introduction of the malady into Brazil in 1849 occurred, it is said, through an infected vessel from New Orleans which landed at Bahia. Before this time, according to some authorities, the disease was unknown in the latter place.

However, others claim that Yellow Fever was imported into Lisbon from Brazil as early as 1723, and that this was its first appearance in Europe. At any rate, since 1849 Yellow Fever has never been absent from Brazil. The introduction of the disease into Portugal from Brazil by vessels followed in 1850, 1851, and in 1856. Again, there occurred at Saint Nazaire, in 1861, the only important epidemic of Yellow Fever that ever broke out in France, and this also was due to infection from shipboard as follows:—

The ship, *Anne Marie*, sailed from Havana on June 13th. On July 1st, eighteen days after her departure from Havana, two seamen fell ill, and died on the fifth day of their illnesses, of Yellow Fever. The epidemic then spread throughout the crew, and nine of the total number of sixteen were attacked with the disease. The ship then entered the port of St. Nazaire twenty days after the last death, and ten days after the inception of the final case of suspicious illness. As no case of sickness had occurred during the last ten days, and as all were well at the time of arrival, the vessel and crew were not quarantined. The *Anne Marie* lay near the shore, and at her side were moored two other vessels. Her crew were disbanded, and the discharge of the cargo was entrusted to seventeen stevedores, all in good health. Shortly after ten of these sickened and died of Yellow Fever, while six people on the shore who had been in contact with the stevedores also fell ill of the disease. In addition, Yellow Fever broke out on board both of the vessels which had been lying near the *Anne Marie*, and from these ships others became secondarily infected. To make a long story short, the *Anne Marie* infected, primarily and secondarily, seven vessels with Yellow Fever. In all there were 40 cases and 23 deaths.

The epidemic in Italy, at Leghorn, in 1804, also owes its origin to a Spanish ship from Havana. During the

voyage to Cadiz, this ship had lost almost its entire crew from Yellow Fever. The crew was recruited at Cadiz, and eventually arrived after several stoppages at Leghorn, where no quarantine was imposed. Two sick men were taken off the vessel, and carried to an inn in the city. Both of them died a few days later; and, shortly after, twelve other persons at the inn were stricken with the disease. Suspicion was directed against the ship from Havana as the source of the trouble, and sanitary guards were stationed aboard. They also promptly took the fever. The malady extended gradually throughout the city of Leghorn. The figures of this epidemic are very conflicting, it having been variously estimated that from 700 to 1,900 people died of the disease. While the malady was not admitted to be Yellow Fever at the time (probably on account of commercial reasons), Palloni, who had witnessed and studied the epidemic, admitted seventeen years later that it had been really one of Yellow Fever.

I will not weary you with the recital of other instances in which epidemics of Yellow Fever have been carried directly by vessels from one port to another, and often over great distances. One need glance only for a moment at the map, and again at the statistics of the location of Yellow Fever cases in many seaport towns for the past year, to be again impressed by the relationship between maritime commerce and the disease. During the past year, among the cities on the eastern coast of the Western Continent, Port Limon, Progresso, Vera Cruz, Tuxpan, Tampico, Rio de Janeiro, Bahia, Manaos, Pernambuco, Para, and Paramaribo, and on the western, Panama and Guayaquil, have all been frequent sufferers from this scourge. It therefore does not seem improbable that unless extreme precautions are taken in regard to vessels passing from these regions and bound for ports in the far East, that infected ships, and even cases of Yellow Fever, will be conveyed from some of these cities to Honolulu, or even directly to Guam, Hong Kong, and the Philippine Islands. I would remind you that as late as 1865 a sailing vessel arrived at Swansea from Santiago de Cuba; Yellow Fever broke out on board while she was in port, and fifteen persons died. It is well known that it is of not very uncommon occurrence in Italian ports to have vessels arrive from South America with a history of Yellow Fever on board during the voyage. With these examples in view, does not the argument seem reasonable that the same thing might take place in Asiatic ports, owing to the

increased traffic from the endemically-infected regions directly to the East? The distance from Panama to Honolulu is 4,692 miles, which is somewhat shorter than from Bahia to Naples, viz., 4,488 miles, and considerably less than that from Rio de Janeiro to Naples, 5,188 miles. Mail steamers leaving Panama should reach Honolulu in from thirteen to fourteen days, or, without stopping, the Philippines in about twenty-eight or twenty-nine days. Vessels, even though leaving Panama with no cases of Yellow Fever on board, and reaching Honolulu with still no history of sickness *en route*, yet might obviously be agents of most serious infection to the Hawaiian Islands: for if infected *stegomyia*, which had had recently bitten Yellow Fever cases, were taken on board the ship at Panama (as the late Dr. Walter Reed has shown), they would not be capable of conveying the disease until about twelve days later, which would be at the earliest time about that of the arrival of the ship in Honolulu. Stevedores from Honolulu, sent on board to unload cargo, might be bitten by such infected *stegomyia*, and could in time convey the disease later to others in the Hawaiian Islands. The same might occur in Guam or in the Philippines; for even though ships should also arrive at these ports with no Yellow Fever on board, and no history of the disease *en route*, yet they might also still contain infected *stegomyia*, which, concealed in the hold, or in other places unfrequented by the passengers or crew, had not had the opportunity of feeding upon susceptible individuals during the voyage. H. R. Carter has reported six such instances. On these occasions, Yellow Fever appeared respectively on the 64th, 68th, 12th, 11th and 38th days after the ship left the infected area. As it has been shown that the *stegomyia* is capable of conveying the disease at least as late as fifty-seven days after biting, it would still be dangerous for many days after the arrival of the vessel in the Eastern port, either if it remained on board or escaped to the land.

The question of whether the *stegomyia fasciata* could be kept alive during a voyage of such duration is easily answered in the affirmative. Reed, however, pointed out that free access to water is necessary for the existence of this mosquito, and that when deprived of it, it dies in from five to six days. There are many instances of mosquitoes being carried great distances at sea, and the *stegomyia* is frequently found on board ships in tropical ports. Dr. Cummings, of the United States Marine Hospital Service, reported that a Spanish barque, sixty-five days from Rio de

Janeiro, arrived at the South Atlantic Quarantine Squadron carrying a veritable plague of mosquitoes, most all of which were the *stegomyia fasciata.* According to the captain's statement, the mosquitoes had been present on board the ship only forty-three days. In the water-tanks of this vessel great quantities of the larvæ were found, showing that the mosquitoes were propagating and multiplying on board. I have purposely cited at some length above two instances in which Yellow Fever was carried in vessels over great distances, and for long periods of time. In the first instance, the ship *Anne Marie* carried the infection from Havana to Saint Nazaire. It will be remembered that the vessel entered the port ten days after the last case of Yellow Fever, and that all on board were at the time well. The infections of the stevedores and others immediately following must have been from the infected *stegomyia* on board the vessel. The history of this epidemic has been carefully explained in this light by H. de Gousea, in *Le Bulletin Medical de Paris,* October 12th, 1901. While it may be argued that as Yellow Fever has been successfully kept out of Italy since 1883, no cases having been registered on shore since this date, even though cases of the disease frequently arrive at Italian ports, that probably the question of its exclusion from our coast cities in the Far East will be an equally simple one. We must remember, however, that evidently the climatic and hygienic conditions in Europe are not favourable for the development of this disease, and that, moreover, the ports I have particularly mentioned—viz., Honolulu, Guam, and the Philippine Islands—all lie within from 10 deg. to 22 deg. north latitude, while the Italian ports are almost all above 40 deg. north. Again, with a greatly increased maritime traffic between the endemic Yellow Fever ports and the East, the danger becomes multiplied manifold, and with each ship so *en route* is the risk further increased. Should, moreover, a case of Yellow Fever, or even infected *stegomyia,* reach Manila without quarantine, the chances would seem to be in favour of its not falling upon barren soil. Humidity and heat, which seem to be, perhaps, the ideal coefficients for the preservation of the disease, are always present in the Philippine ports. Also, the *stegomyia fasciatus* is to be found abundantly throughout these islands, and one may always obtain readily, in a few minutes, a number from any of the dwelling-houses in Manila. Again, in the Philippine ports there will be no on-coming of cold weather to limit the spread of the epidemic, the factor which seems to have

been of most importance in the disappearance of the disease at various times hitherto in the United States. As the *stegomyia,* as well as the conditions favourable for its propagation and development, are to be found everywhere the year round, so an epidemic once started there with a people of perhaps no marked immunity to the disease, would probably only end when no further susceptible material existed.

But while it has seemed wise to emphasise the great importance to these, and its greater risk of infection with Yellow Fever upon the opening of the Panama Canal, in the light of recent results the matter has altogether not a dark side. For example, Wood and Gorgas, stimulated by the work of the late Walter Reed, have recently shown us that it is possible and practicable, by proper sanitation, to entirely stamp out Yellow Fever from its natural endemic home—from a place (Havana) of which, as Dr. Manson himself, writing in 1898, has said: "The disease is never absent." Having shown that this extermination of the malady is possible in such regions, why, then, should not the same be accomplished along the entire route of the Panama Canal? It does not seem too much to hope that this will be brought about, and that before the opening of the Canal there will be little danger of infection from its shores, or from its port of entrance or exit. The measures adopted in the main are to be those already employed by the sanitary brigades in Cuba. Obviously, however, this will not exclude infection from other Yellow Fever ports, such as, perhaps, in Brazil, the West Indies, and Mexico. Over vessels coming from these regions there must naturally be the greatest care exercised, both at the Canal and upon their arrival at the eastern city. The careful work of Dr. Reed has shown that the disease is not conveyed by clothing, sheets, blankets, etc., and that quarantine efforts directed against the transmission of the disease by *fomites* are unnecessary. Moreover, Reed pointed out that infected insects were not likely to be imported in boxes or in trunks, for the reason that when deprived of water the *stegomyia* dies within five or six days. Grubbs, working upon *culex pungens,* arrived at practically the same results, and concluded that these latter mosquitos would not live over six or eight days in trunks and boxes packed loosely with damp clothes. In blankets, etc., they lived a much shorter time, viz., not over two days; all the other carefully-obtained evidence in regard to the conveyance of the disease by baggage seems also to be negative.

In 13 cases of experimental Yellow Fever, Reed and the Army Commission showed that the incubation period varied from forty-one hours to five days and seventeen hours. This also agrees with the incubation period, as clinically observed in a large number of cases by various obervers. A quarantine of an individual beyond six days at any time would therefore be unnecessary.

As to the practical measures that should be adopted at the port of Manila, were the Panama Canal opened under the present existing conditions in regard to Yellow Fever in the West Indies, Mexico, Central and South America, perhaps the safest rule would be that all vessels for Manila arriving from Panama should be sent to the United States Quarantine Station at Marivales. There, all passengers going on shore should be placed at the station for five days in quarantine, and carefully protected from mosquitoes during this period: the great risk involved would perhaps warrant such extreme measures. This proceeding, I may add, is a perfectly practical one, and is scarcely any severer than the quarantine which all our passengers on transports have been compelled to undergo during the recent cholera epidemic. As for the ships themselves, after disinfection as complete as possible against mosquitoes, they should be then allowed to proceed on their way. Unfortunately, as experience has shown, it is not always practicable to destroy all mosquitoes on board, but by quarantine of the ship for a longer period nothing might be gained; and in other ports, nearer infected regions, if *stegomyia* which had recently bitten Yellow Fever cases were still left on board, no case of Yellow Fever would be likely to develop before twelve or fourteen days, and none might develop for a much longer period. In regard to ships which pass through the Canal, and which do not anchor within 200 fathoms of infected places, it seems no quarantine should be imposed. For while, in so far as I know, the exact distance which the *stegomyia* may fly, or be carried by the wind, has not been determined, that of 200 fathoms has practically proved to be a safe one.

I trust that the reiteration of these familiar facts which I have brought before you this evening has not been too tiresome. It has been thought advisable to put the matter in a simple light, in order that the layman may also thoroughly understand and appreciate its importance.

DR. GEORGE H. F. NUTTALL (Cambridge) said: It is difficult to add anything to the excellent and most sug-

gestive paper which Dr. Manson has read. His communication deserves serious consideration.

We are dealing with a broad biological problem, when studying the dissemination of disease. It is well known to biologists that a change in the geographical distribution of the *fauna* and *flora* of the world is rapidly taking place, chiefly through the agency of man; and that animals and plants introduced into foreign countries take their parasites with them. It has again and again been observed that indigenous forms may disappear in the presence of those which have been imported. Assuming even that the distribution of the genus *Stegomyia* were more restricted than appears from the maps of the world which Dr. Manson has shown us, there is every reason to suppose that these insects may become more disseminated, and thus, under favourable conditions serve to maintain Yellow Fever elsewhere. We know that *Culicidae* may survive for weeks on ship-board, and consequently that they may be distributed to distant parts.

The increase of trade throughout the world, as also the rapid communication between ports, made possible by modern ships, greatly facilitates the dissemination of epidemic diseases and their causative agents. Formerly, such epidemic diseases as plague progressed slowly along the old caravan routes—now plague and cholera are scattered broadcast over the world through rapid steamer traffic. There is no reason to suppose that Yellow Fever and other diseases may not pursue the same course.

Those of us who have not been in countries which have suffered from the scourge of Yellow Fever have been thoroughly impressed with the enormous importance of the disease, not only with regard to its fatal effects on life, but also the disastrous influences it exerts on trade and commerce. To accomplish anything along the lines suggested by Dr. Manson, we must impress those in authority with the extent of the losses which might follow the introduction of Yellow Fever into Asia, with its teeming populations existing under conditions eminently favourable for the establishment of the disease in endemic form.

The cost of preventive measures would be infinitesimal, as compared to the financial losses which might follow their neglect. As *an example of the loss of life* due to Yellow Fever alone, in a single city, I would state that it has been estimated that thirty-six thousand people died in Havana in less than fifty years (ending 1900). As *an example of the financial losses* entailed by a single city

from one epidemic alone, I will state that the epidemic of 1878 cost the City of New Orleans a loss of over £2,000,000.

The extent of the losses amongst the Spanish troops in Cuba will probably never be known, but some conception thereof may be formed when I state that I was told the loss amounted to no less than 25 per cent. This was in 1892, when I visited Cuba, my informant being the physician in charge of the military hospital. One ward, set aside for the Yellow Fever season, was styled the "mortuary," the physician in charge thereof the "undertaker," in grim allusion to the fact that the patients who entered the ward rarely, if ever, left it again alive.

DR. SAMBON said: Dr. Manson deserves great praise for his momentous and stirring address. The danger of the wider spread of Yellow Fever is most imminent. Indeed, if I am well informed, its urgency is even greater than represented. The China Commercial Steamship Company, which was incorporated in Hong-Kong last November, will inaugurate on March 1st a service between China and Mexico. The company's steamers will carry both freight and passengers, and a monthly service will be maintained between Hong-Kong and the Mexican ports. Thus, quite independently of the opening of the Panama Canal, within a few days the Asiatic Coast will be in direct communication with one of the great endemic centres of Yellow Fever. A few weeks later, the annual epidemic will break out within the endemic stations of the disease, to become more or less threatening under the influence of the peculiar meteorological conditions of the year, and of other no less important shifts of the animate environment.

Some years ago, we should have been in a terrible plight. Now, although the primary causative agent of Yellow Fever is still unknown, possibly on account of its minuteness, or because it escapes detection within the *leucocytes* or other structures, yet the main epidemiological factors of the disease have been thoroughly worked out by our learned and brilliant American colleagues. The endless and hopeless disputation between contagionists and non-contagionists has at length given way to scientific research; and already we have a sufficient amount of exact knowledge on which to base a sound and efficient prophylaxis against Yellow Fever.

But, though we may well agree as to the means of combating an epidemic of Yellow Fever, I fear that some may

consider Dr. Manson's move as unnecessarily alarming. Only the other day, having mentioned the subject to a medical acquaintance of great influence and authority, I was reminded that Yellow Fever had been known to Europeans since the days of Columbus; that communications between Europe and America had been constantly increasing and improving during the last four centuries; but that the disease had not moved from its endemic region. Although its "germs" had been frequently imported into Europe and North America, Yellow Fever had not gained a foothold in either country; and, being essentially a coast-line disease, it could never become diffused like other exotic pestilences, such as cholera and plague.

The truth is that the distribution of Yellow Fever has slowly but steadily increased. It is only quite recently that the disease has become endemic on the Atlantic and Pacific coasts of South America, and now we see it spreading, by means of its propagating agent, from the coast to the interior, not only through river traffic, but along railways, and regardless of elevation.

Chiefly on account of great engineering changes in the large sea-ports of the Atlantic coast, the United States have been spared, of late years, those frightful epidemics of Yellow Fever which recurred so frequently in the latter part of the eighteenth century and the beginning of the nineteenth. On the other hand, the increasing commercial relations between the United States and Mexico, by reason of the marvellous development which has taken place in the latter country during the last two decades, and the consequent activity in railroad building, are opening up a new and far more dangerous route for the importation of the dread disease.

It is true that in most cases of importation abroad, Yellow Fever did not spread beyond the shipping and wharves, or, at any rate, did not extend beyond the precincts of the town into which it was imported; but we know also from various instances—both in the United States and in Europe—that, given certain conditions, it can spread very widely, and give rise to a deadliness unequalled by any other disease.

Recent advance in the etiology of disease has enabled us to grasp the principles of disease distribution. We know that diseases are always in some way or another caused by living organisms, and that, in many cases, these pathogenetic organisms are fostered and propagated by other living organisms. It is obvious, therefore, that the distribution of

diseases is governed by the same laws which govern all living organisms.

Like other species innocuous to man, so our pathogenetic organisms must have originated within narrow limits, and subsequently spread more widely. Of the spread of many diseases we have no record whatsover; of others we know that they have extended their geographical range considerably within recent times. I need but quote small-pox, scarlet fever, beri-beri, and the chigoe pest.

The present restricted range of certain diseases, and the fact that they have not spread out of their habitat for centuries, do not prove in the least that they are unable to spread more widely. In studying the distribution of insects and fungi injurious to agriculture (that is to say, the diseases of plants), we find numerous examples of species which, although repeatedly imported to certain countries for hundreds of years, have only quite recently established themselves in these countries.

It is not always easy to understand why a certain species will not spread, though frequently imported into a new country. Thus, we do not know why, amongst butterflies, the well-known Garden-white (*Pieris rapae*) should have spread far and wide in America, while the equally common and injurious Cabbage-white (*P. brassicae*) has never been found there. A similar example is offered by the *Filariidae*. While *Filaria bancrofti* has spread very widely throughout the tropical and sub-tropical world, *F. loa* has remained limited to small areas on the west coast of Africa; and, though frequently introduced with Negro slaves into the West Indies, has never become acclimatised in those islands.

At one time, these peculiarities of distribution were easily dismissed by the theory of an uncongenial climate. Now, we know that adaptation to a new country depends upon a number of complex ecological factors. I can do no better than quote Darwin's famous example, by which he showed that in a measure cats are responsible for the production of clover seed in this country, through the inter-relations of cats, field mice, and bumble-bees. Red clover would not grow in New Zealand until bumble-bees were introduced to fertilise its flowers; now it displaces the native grasses.

But whatever may be the obstacles to the establishment of foreign importations, it is undoubtedly true that our rapidly-increasing international trade is the most important factor in the spread of injurious insects, noxious weeds, and disease organisms. Every improvement in rapidity of

travel, and in safety of carriage of goods of all kinds, increases the opportunities of accidental importations.

The commerce in living plants and nursery stock, which has of late years assumed considerable proportions, has afforded a new and most efficient means for the accidental transmission and subsequent establishment of numerous kinds of insects, acari, and fungi. It is by means of importations of this kind that many scale-insects (*Coccidae*), which have usually a rather restricted distribution, have become almost cosmopolitan. Fortunately, the enclosed hymenopterous parasites of these pests have spread almost equally with their hosts.

To take an example from the very region which now concerns us, I will mention the Mexican cotton-boll weevil, which, introduced quite recently in Texas cotton-fields, has proved a serious lesson to the planters of that State.

It is quite obvious that the establishment of a direct and rapid line of transit between Mexico and China will expose the Eurasian Continent to frequent importations of Yellow Fever; and you know full well that we have there the conditions best suited to the widespread and possible establishment of the disease: a wide distribution of the necessary intermediate host, and a non-immune, careless, ignorant, overcrowded, and unruly population.

Our knowledge of the distribution of *Stegomaya fasciata* is still very imperfect, but we know that this truly cosmopolitan mosquito is very prevalent in Southern China, Japan, Siam, India, and Malayia. Another mosquito of the same genus and of similar habits, *S. scutellaris*, is also very common and widely distributed in Asia. It is not unreasonable to surmise that the latter species may also prove a suitable host for the Yellow Fever parasite, since we know that various species of the genus *Anopheles* can subserve the propagation of the malarial parasites.

An outbreak of Yellow Fever in China would probably be followed by epidemics in Japan, Siam, India, or Malayia; and from these countries the disease might well spread to Australia and, possibly, to Europe, along the Red Sea route of many a cholera epidemic.

The prophylaxis against Yellow Fever offers no great difficulty. The means of prevention, suggested by recent discoveries and the experience of a hundred epidemics, are simple, inexpensive, and efficient. But there is a difficulty—an almost insuperable difficulty—and that is the almost universal ignorance and unpardonable neglectfulness of those whose bounden duty it is to forsee any possible

danger and endeavour to avert it. Truly, the Ancients were right when they stated that epidemics were the chastisement of sin!

I trust that on this occasion the authorities will take warning, and thus avoid the horrors and humiliation of a Yellow Fever epidemic in Asia. That the risk is great no one can reasonably doubt; but, at the same time, we know for certain that it can be prevented. Anyhow, whatever may be the course of events, we can assert that physicians have done their duty; many have yielded their lives to save mankind from this fearful monster of the Western Hemisphere.

I will not waste any time in describing minutely the measures to be adopted in order to prevent or repress an outbreak of Yellow Fever. The cleansing of Havana is already more famous than that of the Stables of Augeas. The admirable measures adopted by Major Gorgas, of the United States Army, were based upon the recently-acquired knowledge that Yellow Fever is propagated by mosquitos, and by mosquitos only.

In order to prevent the further extension of Yellow Fever, the most important step is the extirpation of the disease from its endemic stations. So long as there is Yellow Fever in Central America, in South America, and in the West Indies, the United States and Eurasia will run the risk of its annual invasions. I think that Great Britain and the United States should co-operate for the enforcement of the measures necessary for the removal of this danger.

It should be the duty of Governments possessing threatened sea-ports to carry out such engineering changes, and other appropriate measures, as would tend to wipe out the offending mosquito.

All vessels leaving suspected districts should be thoroughly fumigated on departure, and again on arrival, whether Yellow Fever has broken out on board or not. The possibility of carrying mosquito larvæ should be guarded against by frequent inspection of water-tanks.

Captains and officers of ships should be thoroughly acquainted with the *rôle* played by the mosquito, and with the measures necessary for the prevention of Yellow Fever. Any disregard or carelessness on their part should be heavily fined.

Any idea of establishing a quarantine should be unhesitatingly set aside. Quarantines hamper commerce, entail useless expense, and encourage a fatal disposition

to conceal infection. The first practical requirement towards checking the spread of a disease lies in its prompt detection; therefore, notification should not entail any unnecessary inconvenience, and should be insisted upon with the utmost severity. Experience has proved again and again that the Yellow Fever patient, screened from the bite of mosquitos, is of no danger whatever to the community.

Gentlemen, twenty-four years ago Dr. Manson discovered that mosquitos were the intermediary hosts and propagators of filarial infection. Since that day, a new generation has sprung up. Those who have visited the Tropics know how fearfully this new generation has been visited by *Filaria bancrofti* and its attendant horror, *Elephantiasis*. Only consider how much wretchedness might have been avoided had Dr. Manson's discovery been acted upon!

Alas! even Erostratus has followers!

Dr. G. C. Low said: I have not very much to say on the subject under discussion; but from my own practical experience of the disease I can confirm the great danger of its introduction into new areas. Dr. Strong has given you many instances of its spread by mosquitos escaping from infected ships, and a similar instance to some of these has been investigated by myself in St. Lucia.

Ships and steamers had been in the habit of coming from Rio and other Yellow Fever ports in South America to coal at this island; and from one of these mosquitos escaped, and a white sergeant of police in the town was evidently bitten, and developed Yellow Fever. On the first day of his illness, when his premonitory symptoms were developing, he spent a portion of his time in the sergeant's mess on the Morne, the military station. More mosquitos bit him there, it must be assumed, became infected, and bit other people. Yellow Fever subsequently broke out amongst the troops. The prompt action of the military authorities in changing the troops to another site prevented a serious epidemic, and the mosquitos in the affected areas were killed by destroying their breeding grounds, and by fumigating them in the houses. The military authorities followed out the suggestions made and improved their areas permanently, but the town of Castries did not stir in the matter at all. The point I wish, however, to specially point out is, what practical steps are to be taken to stamp out mosquitos and so prevent the spread and the continual danger of the introduction of this disease. The scientific world is agreed on the point that the mosquito spreads

Yellow Fever, malaria, and filariasis; yet, even after this has been proved, what has the English Government done to take practical steps to prevent those diseases? I am sorry to say, practically nothing. Major Ross, with private funds, has tried to show what can be done in Sierra Leone, and the War Office has also been at work to perfect the military areas there; but it is unreasonable to expect enthusiasts to privately clean up and improve the sanitation of all the tropical towns in the world. While in St. Lucia I proposed a useful scheme to improve the sanitation of Castries, the main town, which is a standing disgrace to all sanitary laws; but the administrator was lukewarm on the subject, and nothing has been done. Again, in Barbados, I showed how easily mosquitos could be stamped out, and the enormous prevalence of filariasis diminished by simply passing a law about people keeping standing water in their houses. Though Sir Frederick Hodgson, the Governor, took the greatest interest in this scheme, and helped me in every way, what was the result? The local parliament decided not to pass the law, and the mosquito reigns supreme. It is this indolence and indifference in British Colonies that is so aggravating. Why should they not have a part of their annual income or grant set apart solely for the purpose of sanitation? Unless the Government take action in this matter of the Panama Canal, and the introduction of Yellow Fever into Asia, exactly the same thing will occur, namely: the matter will be treated with laziness and indifference; captains of ships will pooh-pooh the idea, not fumigate their ships, and after, when the disease has been introduced (that is, when it is too late), people will begin to talk about doing something. With the example of what Italy and America has done in regard to mosquito hygiene before us, it is nothing short of a disgrace that we should have done so little. The mosquito theory has passed away; it is now no longer a theory but an actual fact that they spread those diseases, and acting practically, not talking, is what is now required.

DR. EDWARD HENDERSON (late of Shanghai, China) said: I have listened with much interest to Dr. Manson's able address, as I am sure we have all done, and to the interesting comments which have followed it from the various speakers. The danger pointed out—that of the introduction of Yellow Fever to Asia and the Far East—by the construction and opening up of the Panama Canal—is, so far as I am aware, not one which has

as yet attracted that degree of public attention which it should have done. That it is a real danger, and one which should receive immediate and serious attention, there can, however, be little doubt. The view taken of the position by Dr. Manson is by no means one founded on theoretical considerations—that would be a comparatively poor basis, and one on which few Governments would care to take any decided action. It rests, on the contrary, on well-ascertained facts, and on facts established in the surest way—the way of scientific experiment and accumulated experiences. We know the history of Yellow Fever, its malignancy, its highly contagious character, and its easy transport to a distance from its endemic area: and we also know the conditions which favour its rapid extension, and are essential to its endemic continuance. In addition to all this, we have, within the last few years, acquired the knowledge, thanks to the labour and devotion of American men of science in Cuba and elsewhere, that a special mosquito is the most active—possibly the sole—agent in its distribution.

I say, definitely, we have "acquired this knowledge," because the necessary investigations have been so carefully, so scientifically, and so completely carried out, that it seems to me impossible to doubt the accuracy of the results arrived at. With all this knowledge it would be strange indeed if we could regard with indifference the opening up of a new and greatly shortened trade route to Asia and the Far East, which will pass directly through the area in which Yellow Fever is endemic. Panama itself has been described as the hotbed of Yellow Fever; and in the West Indian islands, through which the route is continued eastwards, Yellow Fever is always more or less present.

I have myself been engaged in active practice in Shanghai for now over thirty years; and during that time, for over twenty years, I have held the position of Medical Adviser to the Municipal Council, which presides over the British division of the settlement. As the result of this extended experience, I am only too familiar with the disastrous consequences which follow when epidemic disease is introduced among the natives of a hot climate; and I cannot contemplate, without grave apprehension, the future of the Shanghai community should Yellow Fever ever be introduced there—a community in which foreigners and natives are, and must continue to be, intimately associated. The dirt and overcrowding which determine the virulence and extension of an epidemic, though possibly

absent in the foreign settlement itself, are never likely to be wanting in the closely-adjoining native city, while the temperature of the Shanghai summer would supply the only other requisite—sustained tropical heat. Quite possibly an outbreak of Yellow Fever in Shanghai might terminate as a single epidemic, for the climate of Shanghai is a sub-tropical one, and Yellow Fever is endemic only in the Tropics. I say an outbreak might terminate as a single epidemic, but think what that means in the case of Yellow Fever; it might mean the death of 10 per cent. of the whole inhabitants—it did mean that once in Philadelphia, when 4,000 died out of a population of 40,000. I have spoken of Shanghai because all my personal associations with China were centred there: but what of the thriving British colony of Hong Kong? Again a mixed community, where foreigners and natives are intimately associated, Hong Kong is in the Tropics, and Yellow Fever once introduced there would find all the conditions necessary for endemic continuance. It might fail to make a lodging in Hong Kong itself, but might easily do so among the natives in Canton or the surrounding district. What I have said of Shanghai and Hong Kong has, of course, a very wide application in the great and populous Empire of China.

Among the various precautionary measures to be taken to avert the possibility of such a calamity as the one we are now considering, the destruction of the special mosquito now known to be the active agent in the dissemination of Yellow Fever, should, as suggested by Dr. Manson, be made the principal object. The details are simple enough: such details as were carried out with the happiest result by the American Government in the city of Havana within the last three years: details differing in no essential particular from the arrangements made by Major Ross and his co-workers in dealing with malaria. Success will ultimately depend on the ability of the Government undertaking the task, to secure the services of a staff sufficient in numbers and intelligence to carry out the work thoroughly. And I am glad to leave the further discussion of these measures in abler hands than my own. Nothing must be left to chance in so important a matter—a matter which concerns the lives of thousands, and may one day seriously affect the vast trade interests represented in China and the Far East.

After a few remarks by SIR WILLIAM KYNSEY, C.M.G., late Principal Civil Medical Officer and Inspector-General of Hospitals in Ceylon,

DR. SIMPSON said, that Dr. Manson had very clearly set forth the dangers likely to follow the opening up of a new trade route by the construction of the Panama Canal. History showed that epidemic diseases followed trade routes. Taking plague as an example, this disease had not only followed the trade routes, but had died out in those countries in which it had prevailed where the trade routes were changed. For instance, the disappearance of plague in Europe, in the latter part of the seventeenth century, was due in a large measure to the change of trade routes brought about by the discovery of America and the West Indies, and the new sea route to India *viâ* the Cape. He agreed with Dr. Manson that quarantine, or for that matter any other precautionary measure to be applied at the terminal ports in the East, was not likely to be successful in keeping out Yellow Fever. China would never carry out any sanitary measure. Precautionary measures would have to be taken at the ports of departure. Who should be the authority to carry out these measures would be a matter for consideration. The question was an international one; more countries than America and Great Britain were concerned in the threatened danger. There were also the Dutch and the French, whose tropical colonies would be affected. It would therefore have to be considered from an international point of view.

INSPECTOR-GENERAL ALEXANDER TURNBULL, M.D., R.N., said: At the Edinburgh Meeting of the British Medical Association in 1898, I read a paper on "Insanitary Environment the Cause of the Spread of Yellow Fever." The exciting cause of this disease was not then apparent; the discovery of the part the mosquito plays in propagating the disease is of a later date.

My difficulty at first in accepting this now-established fact was the apparent occurrence, on October 7th, 1845, of Yellow Fever in a pilot, who had embarked in the *Eclair*, a Yellow Fever ship, at the Motherbank, on October 1st, and died on the 10th, at Sandgate, at the mouth of the Thames. It is, however, recorded that, in his case, "the head appeared to bear the onus of the illness." With the present accurate knowledge of the incubation stage of Yellow Fever, it may be concluded that this was not true Yellow Fever. "He was exceedingly alarmed from the first;" and, it may here be stated, that though two Yellow Fever patients were landed and died with suspicion of black vomit, in the Marine Infirmary, Wool-

wich, there was no extension of the disease to their attendants.

On January 15th, 1870, at Rio de Janeiro, I assisted in diagnosing a case of Yellow Fever, soon quickly fatal with black vomit, on board H.M.S. *Egmont*, in a close, unventilated dock; the ship was at once moved to a more open anchorage, and no further case occurred until the 30th.

In 1871, I was at Buenos Ayres on the outbreak of what proved a decimating epidemic of Yellow Fever in that city, spreading up the Parana and Paraguay rivers, in the towns on their banks, but having no extension to the open country adjoining of the same level—save in an exceptional case or two, possibly—though the sick moved to this new ground died as in the towns.

Rio de Janeiro, in the "Forties," was a sanatorium for West Coast of Africa ships' crews; ere long Yellow Fever was introduced there, and is now endemic. All these cases are explained by mosquito inoculation, amid insanitary environment.

The following authentic official particulars appear germane to the subject under consideration.

H.M.S. *Eclair* developed Yellow Fever amongst her crew in July, 1845, at Sierra Leone; in August she introduced it into Boa Vista; and, by December 22nd, two hundred and fifty natives had died of it; by March, 1846, the deaths numbered four hundred.

In 1865, a working party from H.M.S. *Bristol*, a fine, airy frigate, were employed on board a stationary storeship at Sierra Leone. Yellow Fever broke out among this party, on board the *Bristol*, viz., on their return to that ship; they were treated on her airy main deck, and no other cases resulted in the *Bristol*, unlike the *Eclair*, on board of which ship the disease increased all the way to England, the last fatality being in the case of a Lieutenant, who had been on board the ship from the onset of the fever, in close attendance on the sick; he was siezed on October 7th, at Sandgate, at the mouth of the Thames, nine days after reaching England.

The mosquito inoculation will account for his fever, in an ill-ventilated paddle-steam sloop, and the immunity of the *Bristol's* crew is explained by the sick being treated on her airy main deck.

A question, however, that arises in connection with these two Sierra Leone infected ships is, If Yellow Fever is now endemic at that tropical spot, and commerce affected thereby?

H.M.S. *Dauntless*, November, 1852, landed 62 cases of Yellow Fever at Barbadoes; they were treated "indiscriminately" with other patients in the military hospital.

H.M.S. *Highflyer*, December, 1852, sent 51 cases to the Naval Hospital at Port Royal, Jamaica, who were "mingled" with other sick there.

H.M.S. *Esk*, in November, 1853, landed 8 cases at Nassau, with black vomit.

In none of these three instances did the disease spread in the islands; though, in the *Dauntless*, it did in the ship anchored at Barbadoes.

There is no Yellow Fever in the River Plate or its affluents, the Parana and Paraguay, now, I believe.

At the conclusion of the discussion, a hearty vote of thanks was accorded to Dr. Manson for his valuable contribution, and it was resolved to forthwith print the paper and the discussion thereon.

It was also further resolved that a Committee, consisting of the President of the Society (Professor Corfield), Dr. Cantlie, Sir William Kynsey, C.M.G., Sir Francis Lovell, and Dr. Nuttall, should be appointed to consider and determine what action should be taken upon Dr. Manson's paper, and to report to the Society the result of their deliberations.

NOTE ON THE GENUS "STEGOMYIA" (THEOBALD), AND ITS DISTRIBUTION.

By F. V. THEOBALD, Esq., M.A., F.L.S.

The genus *stegomyia* ranks next to *anopheles* and the related genera of *anophelina*, on account of the part it plays in the distribution of Yellow Fever. How many of the species in this genus may be implicated we do not at present know; at present a single species only seems to be held responsible. That species (*S. fasciata*), is one of the two commonest West Indian and South American *culicids*. Although *fasciata* is widely distributed in all tropical and sub-tropical countries, it does not occur in any numbers in certain parts, such as the Malay States, China, and in Africa. Where it does not occur in abundance it is nevertheless represented by closely-allied members of the genus, such as *scutellaris* in the Malay States, China, etc., and *Africana* and *argenteo punctata* and others in Africa. It is quite possible that the latter species and the common

Australian *notoscripta* might play the same *rôle* as *fasciata*, given a sufficient Yellow-Fever grazing ground. The genus seem to have a decided preference to the littoral. The genus was separated from *culex*, entirely on account of the scale structure of the head, thorax, and wings; and, as has been recently shown, there is as much structural difference in the larvæ of the two genera. In regard to the eggs (if records are correct) there are, however, discrepancies, for *fasciata* and *scutellaris* lay their eggs singly, but, according to Skuse, those of *notoscripta* are deposited in "rafts." Little is known, however, of the life-history of any members of this genus except in *fasciata*.

From information I have been able to collect they all seem to be vicious bloodsuckers, and this habit does not only seem to be confined to the females. The reports of the males being bloodsuckers requires further investigation, however. These insects bite both by day and night, and, in all, the bite alone is most annoying, irrespective of the insects possibly carrying Yellow Fever. It is in this genus that we find special predilection for settling, when at rest, on dark objects and clothing. They have been popularly known as "Tiger-mosquitoes," on account of their banded and striped appearance; but a glance at the synoptic Table will show that that name is misleading, as many members of the genus are unbanded or unstriped. They are, however, mostly small dark gnats, with white, silvery, or yellow lines, bands, or spots on the thorax and legs (*fasciata, sugens*, etc.), or they may be unadorned (*brevipalpis, nigricephalae*, etc.). The largest species is *cranipes* (4.8 to 5 in.), an aberrant form of a more general brown hue than the rest, and *grantii*, which is often as large; but both can at once be told by the flat head and scutellar scales. They mostly seem to be more or less connected with man, some being exclusively domestic, others only partially so.

Certain species (as *fasciata, scutellaris, notoscripta*, etc.) are found not only in houses, stables and sheds, but also on ships and trains, and are doubtless distributed with the *culex fatigans* in that manner.

Longevity of Adults and Eggs.—Through the kindness of Dr. Finlay I received, last year, some ova of *fasciata* from Cuba. They were sent dry in a tube, and were left for two months after arrival. As an experiment they were placed in some tepid water in my greenhouse, and all the fifty eggs hatched within twenty-four hours, and from these several adults hatched out. Further, these adults were

kept for two months without food of any kind, but with moisture, and at the end of that time, not having been supplied with water, they died during my absence from home.

Pairing of S. fasciata takes place in sunshine; the male invariably gets under the ♀, who may or may not carry him off and complete the process in the air. The males fertilise a large number of ♀'s, never tiring according to Dr. St. George Grey's observations.

The genus is certainly a remarkable one in many respects, but most of all in regard to the wide distribution of the type species, *S. fasciata.* The original home of this species is undoubtedly the West Indies and the north of South America, where it is most abundant, most vicious, and of greatest importance in regard to the part it plays in the dissemination of Yellow Fever. Fortunately, being an almost exclusive tub and pot breeder, it can be more easily dealt with than the various *Anophelina,* which have wider breeding grounds.

At present there are twenty-two species described: I have seen all but one of these (*signifer Coquillett*). Another new species, too broken to describe, was sent me by Dr. Sergent from Algeria, and one by Dr. Lutz from Brazil, that I have had the misfortune to mislay, so that there are known to really exist twenty-four species, all of which are easily identifiable on account of their striking thoracic and other ornamentation.

The known species may be easily distinguished by the following synoptic Table :—

SYNOPTIC TABLE OF *STEGOMYIA.*

A.—PROBOSCIS BANDED.

1. Thorax, with five silvery and yellow lines ... *Notoscripta.* Skuse.
2. Thorax unadorned; black, with yellow-golden scales ... *Periskelta.* Giles.

B.—PROBOSCIS UNBANDED.

(*a*) *Legs basally pale banded.*

β. Abdomen banded basally.

3. Thorax with momedian yellow lines and lateral silvery curved lines; ungus of ♀ serrated ... ... *Fasciata.* Fabr.
4. Thorax as in 3; ungus of ♀ simple ... ... *Signifer.* Coquill.
5. Thorax, with two short median pale lines in front, and a pale spot on each side of them ... ... *Nigeria.* Theob.
6. Thorax, with narrow median white line forked in front of scutellum, a fine curved lateral pair, and a short pure white one below on each side .. ... *Grantii.* Theob.

7. Thorax, with momedian yellow lines and two pale creamy lateral lines in front ... *Sexlineata.* Theob.
8. Thorax, with one silvery median line, and lateral curved lines ... ... *Scutellaris.* Wele.
9. Thorax, with five white spots ... ... *Sugens.* Wied.

ββ. Abdomen unbanded.

10. Thorax, with two pairs of lateral oblique silvery bars ... *Africana.* Theob.
11. Thorax, with a broad patch of white scales on each side in front, and a median pale line ... .. *Terrens.* Wele.

(*aa*) *Legs apically banded.*

12. Thorax, with two prominent and two pallid spots *Marshallii.* Theob.

(*aaa*) *Legs apically and basally banded.*

β. Abdomen with basal white bands.

13. Thorax, with median white line, a large lateral curved one, a small straight white one on each side of median line in front, and another on each side behind ... ... *Pseudo taeniatus.* Giles.

β. Abdomen unbanded, with white lateral spots.

14. Thorax, with round median white spot in front, two lateral white areas, median area prolonged downwards laterally ... ... *Gubernatoris.* Giles.

(*aaaa*) *Legs unbanded.*

β. Thorax adorned.

15. Thorax, with four brilliant white spots ... *Argenteo punctata,* Theob.
16. Thorax, with two pale spots and two median pale lines ... ... *Minuta.* Theob.
17. Thorax, with two dark spots ; head white *Albocephala.* Theob.
18. Thorax, with front half silvery ... ... *Nivea.* Ludlow.

ββ. Thorax unadorned.

19. Abdomen, with basal bands .. .. *Irritans.* Theob.
20. Abdomen, with apical pale bands ... *Crassipes.* V. d. Wulp.
21. Abdomen, with basal lateral white spots ... *Nigricephala.* Theob.
22. Abdomen unbanded ... ... *Brevipalpis.* Giles.

NOTES ON DISTRIBUTION OF SPECIES.

(In nearly all cases the species are found over a wide area around the locality given ; where otherwise, it is mentioned in each species.)

1. *Stegomyia notoscripta.* Skuse.

This species has, so far, only been found in Australia. Specimens have been sent from Queensland, where it seems to be abundant. It is also common in New South Wales, and has been also sent from Victoria and South Australia. It is a household form ; but Dr. Bancroft has found it biting in jungle. The *larvae* breed in water-butts. It is one of the common annoying *Culicids* of Australia. We may expect it to occur elsewhere.

2. *Stegomyia Nigeria.* Theobald.

A single specimen only has occurred. Taken at Bonny, West Africa. Evidently rare.

3. *Stegomyia Grantii.* Theobald.

So far only recorded from Sokotra, where it is abundant and very annoying.

4. *Stegomyia sexlineata.* Theobald.

A very marked species, so far represented by a single ♀ from Trinidad.

5. *Stegomyia scutellaris.* Walker.

A very abundant species in parts of India, Malay States, East Indies, China, Japan, etc. It bites very severely, and occurs in and around man's habitations. It will probably prove to be a Yellow Fever carrier. It has been sent frequently as *fasciata*, but can at once be told by the single silvery median thoracic line.

The following are its recorded localities :—

India.—Ceylon ; Madras ; Canara district, Goa ; Central Provinces ; Naini Tal.

Malay States, etc.—Selangor ; Penang ; Perak ; Singapore ; Siam.

China.—Hongkong ; Foo-chow ; Thaohyling.

East Indies.—Borneo ; Celebes ; Amboina, and British New Guinea.

Other Islands.—Seychelles ; Formosa ; Japan ; Fiji and Mauritius.

6. *Stegomyia fasciata.* Fabr.

The most widely-distributed member of the *genus.* Previously described under a variety of names. Essentially a domestic form, and bites with severity. The male, as well as the female, *said to bite.* This species is found in houses, tents, stables, ships, trains, etc. It is subject to considerable variation in size; but the thoracic markings are constant, except in the variety *mosquito,* in which the two median pale lines on the thorax are almost or quite obliterated. It does not seem to occur further north or south of line 43 deg.

The following are its recorded localities :—

India.—Ceylon ; Travancore ; Madras ; Central Provinces ; Calcutta.

Malay States, etc.—Siam ; Perak. (Apparently rare, and represented by *scutellaris.*)

East Indies.—Celebes and New Guinea.

Palestine.—A few specimens recorded.

Japan.—A single specimen.

Australia.—Queensland ; New South Wales ; Victoria and South Australia. Known here as *Culex Bancroftii.* From the malarious uplands of Victoria it has ueen sent in numbers, but no *Anopheles.*

Africa.—Sierra Leone ; Lagos ; Nigeria; Gambia ; Senegambia ; Old Calabar ; Mashonaland ; Pretoria ; Durban ; Nairobi ; Zanzibar ; Port Said ; Zomba, British Central Africa generally. None have occurred in the large collections from Uganda.

North America.—Savannah ; Georgia, etc. Abundant in Southern parts of the States, and as far North as Atlantic coast at Virginia.

South America.—British Guiana ; British Honduras ; French Guiana ; Demerara ; Panama ; Brazil ; Argentine Republic.

West Indies.—In all the Islands.

Europe and Mediterranean Islands.—South Italy ; Spain ; Portugal ; common at Gibraltar ; Cyprus ; Crete.

Oceanic Islands.—Fiji ; Seychelles ; Mauritius ; Bermuda.

Stegomyia periskeleta. Giles.

The only locality known for this species is Shajahanpur, N.-W. Provinces, India. Evidently rare.

Stegomyia Africana. Theobald.

Fairly common in West Africa. Recorded from Sierra Leone; Lagos; Old Calabar; Zomba, and Mashonaland. (Evidently rare in the two latter places.)

Stegomyia sugens. Wiedemann.

Apparently rare, but widely distributed. Occurs in Sierra Leone; Mashonaland; Nubia, and Corsica.

Stegomyia argenteo punctata. Theobald.

Recorded only from Mashonaland.

Stegomyia minuta. Theobald.

A single specimen known only; from Mashonaland.

Stegomyia albocephala. Theobald.

A single male only known; from Gambia.

Stegomyia nivea. Ludlow.

Apparently limited to the Philippine Islands.

Stegomyia Marshallii. Theobald.

So far recorded only from Mashonaland and Gambia.

Stegomyia pseudo tereniata. Giles.

Occurs in the hills in India, up to 8,000 ft. Bakloh, Punjaub; Naini Tal (7,000 ft.); Himalayas (8,000 ft.)

Stegomyia gubernatoris. Giles.

Represented by a single female from Allahabad, India.

Stegomyia irritans. Theobald.

So far found only at Bonny, West Africa.

Stegomyia crassipes. Van der Wulp.

Burmah and the Indies. No recent records.

Stegomyia brevipalpis. Giles.

Found with *S. piperokeleta* at Shahjahanpur, N.-W. Provinces, India, only.

Stegomyia nigricephala. Theobald.

Recorded only from Bonny, West Africa.

Stegomyia terrens. Walker. South America.

The type only has been recorded.

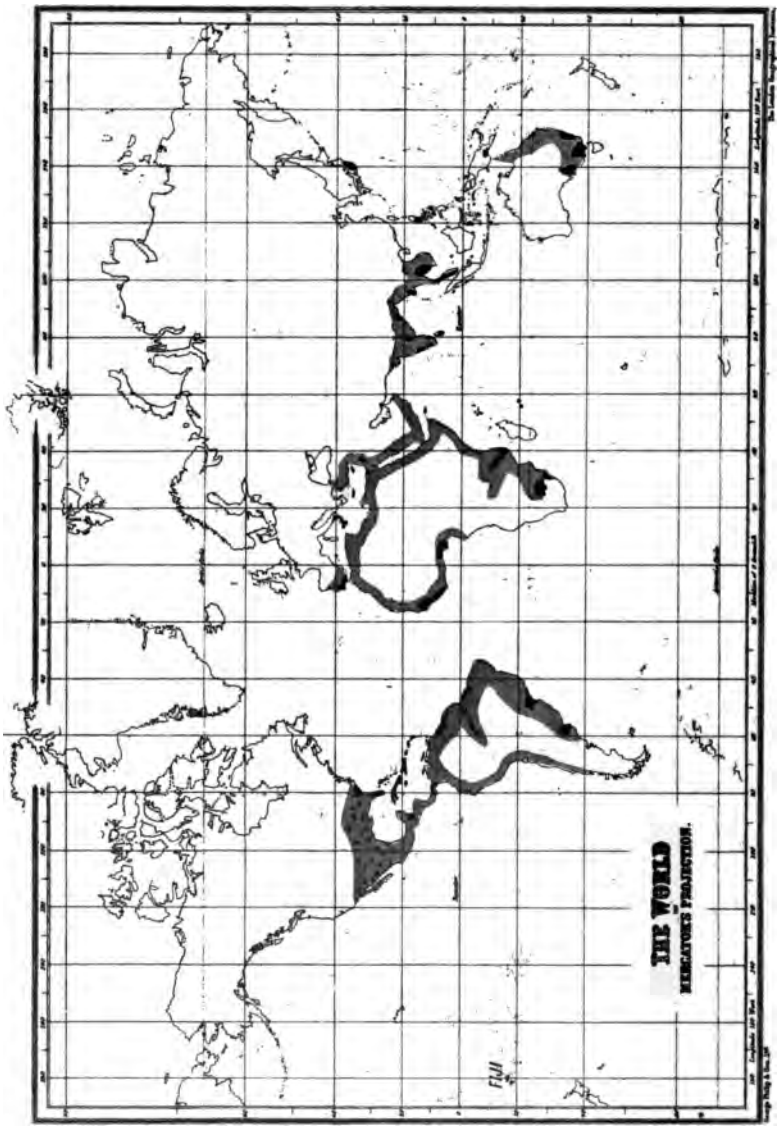
It will thus be seen that, as far as we know at present, only three species are very abundant, namely, *fasciata scutellaris*, and *notoscripta*. The only two species with a wide distribution are *fasciata* and *sugens*.

The *genus* does not occur, as far as we know, outside 43 deg., but we may expect it to occur anywhere within 43 deg. N. and 43 deg. S.

If, of course, *Calopus* (Meigen), is *fasciata* (Fabricius), then it has occurred in England; but I am extremely doubtful upon this point, so many errors have been made in identification and the confusion of synonomy.

The species that seem to be most important after *fasciata*, in regard to "Yellow Fever," are *scutellaris* and *notoscripta*, both of which, being very closely related to the *fasciata*, might prove to be carriers of the disease.

The following maps have been prepared to show the probable distribution of the genus *Stegomyia* in the world.



The Dark Areas show the places from which *Stegomyia fasciata* has been recorded.

WESTERN HEMISPHERE.

DISTRIBUTION OF THE GENUS STEGOMYIA.

1. The Dark Areas show the places from which the genus has been recorded so far.

2. The Pale Areas show where we may expect to find it.

EASTERN HEMISPHERE.

DISTRIBUTION OF THE GENUS STEGOMYIA.

The Dark Areas show the places from which the genus has been recorded so far.

The Pale Areas show where we may expect to find it.

THE PREVENTION OF DIPHTHERIA OUTBREAKS IN HOSPITALS FOR CHILDREN.

By LOUIS PARKES, M.D., D.P.H., Medical Officer of Health for the Metropolitan Borough of Chelsea.

(*Read: March 20th,* 1903.)

In September, 1899, I was asked to advise the Committee of a Children's Hospital in Chelsea as to the measures requisite to prevent continually recurring outbreaks of diphtheria and septic throat amongst the patients and hospital staff. For many years past there had been trouble with diphtheria in this hospital, but during the years 1897, 1898, and 1899, there had been so many outbreaks, involving such comparatively large numbers of the inmates, and the wards had been so frequently closed for disinfection, that the committee viewed the situation with the greatest concern, and some even contemplated the closure of the institution and the abandonment—at any rate temporarily—of its work.

The hospital contains two wards, making up at that time fifty-six beds, and has a staff of about thirty nurses and servants. Briefly, the history of the hospital as regards diphtheria in these three years was as follows: In 1897 there were three distinct outbreaks. No. 1.—Six cases occurred between January 7th and February 13th, the longest interval between any two successive cases being eleven days. There was then an interval of four and a-half weeks without cases. No. 2 commenced on March 16th and continued to May 11th, comprising three septic throats to start with, there being an interval of nineteen days between the second and third cases, and followed by 13 cases of diphtheria, the longest interval between any two successive cases being six days. The hospital was then entirely closed from April 27th to May 24th. On the reopening of the hospital there was an interval of twelve weeks without cases. No. 3 commenced on August 17th and lasted until October 27th, comprising 7 cases of diphtheria and three septic throats. There were intervals of eighteen days, seventeen days, and fourteen days, between

successive cases. From October 27th, 1897, to January 4th, 1898, a period of ten weeks, there were no further cases.

In 1898 there were four distinct outbreaks. No. 1.—Five cases occurred between January 4th and February 8th, the longest interval between any two successive cases being fifteen days. From February 8th to April 14th, a period of nine weeks, the hospital was free from diphtheria, but one or other of the two wards was closed for the greater portion of this period. No. 2.—From April 14th to June 16th there were 5 cases of diphtheria, and four septic throats. There were two intervals of sixteen days and twenty days between successive cases. Each of the two wards was closed for a portion of this period—April 14th to June 16th. There was then an interval of ten weeks, June 16th to August 23rd, without fresh cases. No. 3.—Between August 23rd and September 20th, 4 cases of diphtheria occurred, there being two intervals of eleven days each between successive cases. From September 20th to November 18th, a period of eight and a-half weeks, there was a cessation of fresh cases. No. 4 commenced on November 18th, and continued until February 20th, 1899, comprising 21 cases of diphtheria and eight septic throats: the longest interval between any two successive cases was eighteen days. One of the wards was closed during a portion of this period.

In 1899, consequent upon the preceding outbreak, the entire hospital was closed from March 4th to May 1st. From May 1st to June 20th, a period of seven weeks, there were no fresh cases. On June 20th a fresh series commenced, comprising 10 cases of diphtheria and one septic throat, and lasted until August 9th. In this outbreak there were two intervals of eighteen days and twenty days respectively between successive cases. The hospital was closed on August 4th, and was not reopened until October 26th, 1899, when the recommendations contained in my report to the committee had been given effect to.

During the eight outbreaks above mentioned there had been 65 cases of diphtheria amongst in-patients and 9 cases amongst the staff. There had also been 16 cases of septic throat amongst the staff, which in all probability were diphtheritic in nature, making a grand total of 90 cases. During these outbreaks there had been ten occasions in which the interval between successive cases had been between two and three weeks in duration, these intervals being longer than the known incubation period of diphtheria.

The above narration shows that during the two and a-half years, 1897-8—August 1899, the hospital was never free from throat mischief for a longer period than twelve weeks at a time; that the closure and disinfection of the wards was not effective in keeping the hospital free from attacks, the intervals of freedom being very nearly as long, when the wards had not been antecedently closed, as when they had been. Although in the sequence of cases characterising the outbreaks, there had been many cases which appeared to have acquired the infection directly from antecedent cases, as shown by the short intervals of time elapsing between successive cases, still in a considerable proportion (about 13 per cent.) the known incubation period was much exceeded in the intervals between successive cases. The inference is that in these cases, and possibly in some of the others, some ten in number, where the interval exceeded a week, the disease was either freshly introduced into the wards from some external source, or else an originally dormant source of infection within the hospital became active, and acquired infective properties. All the cases occurring were isolated and removed to the M. A. B. fever hospitals, as soon as a diagnosis was arrived at.

The drainage and general sanitary arrangements of the hospital were satisfactory; but the building, not having been constructed for use as a hospital, there was no proper disconnection by intercepting lobbies of the bath and sink rooms and water-closets from the wards. The general system of cross-ventilation of the wards was good, but certain exit ventilators in the walls and ceilings, not accessible for cleaning, were ordered to be closed, and all the Tobin Tube inlets were made detachable and accessible for periodic cleansing. The number of beds in the hospital was reduced from fifty-six to forty-two, the latter number practically allowing 100 square feet of floor space per bed.

With regard to administration, my chief recommendations were: (1) that stringent precautions should be adopted to prevent the carriage of any infective matter from the out-patient department into the wards; (2) that all discharges and excretions coming from the wards should be disinfected, and that the nursing staff should, as far as possible, disinfect their hands when soiled; (3) *that no patient should have anything introduced into his or her mouth that had not been previously sterilised.* This rule involved the adoption of a system of—after cleansing—placing all spoons, cups, forks, feeding-bottles, teats, feeders, medicine glasses, thermometers and spatulas, in a sterilis-

ing solution, in which they remained until again required for use. The sterilising solution at first tried was a 1 per cent. solution of formalin. This was found to have a very injurious effect on the nurses' hands, causing cracks and sores most difficult to heal. The strength was subsequently reduced to ½ per cent. and then to ¼ per cent., but the unpleasant effects on the skin were not altogether obviated by these weaker solutions. On October 1st, 1900, 1 in 80 carbolic was tried instead of the formalin, and this strength was continued until December 14th, 1900, when an outbreak of 4 cases of diphtheria in the hospital, between the 1st and 12th of that month, having given ground for the belief that 1 in 80 carbolic was not efficient, the strength of 1 in 40 carbolic was introduced, and has been continued to the present time. This has been the only outbreak of diphtheria in the hospital from the reopening in October, 1899, up till the present time. It necessitated the closing of one of the wards for fourteen days. There have been other isolated cases of diphtheria—to be mentioned later; but no other outbreak of consecutive cases, such as that in December, 1900, which resembled the commencement of the outbreaks so frequently seen in 1897 and 1898. In my own mind I attribute this outbreak to the inadequacy of 1 in 80 carbolic to destroy pathogenic throat organisms present in mucus, which may adhere to cups, spoons, etc., even after ordinary processes of cleansing.

The other recommendations contained in my report were: (4) that no patient should be admitted as an in-patient until the throat had been medically examined, all suspicious throat cases being rejected; (5) that swabs should be taken from the throats of all patients on admission, and sent to the Jenner Institute for bacterioscopic examination. This was more or less systematically done from the end of November, 1899, until the end of January, 1902, when it was discontinued. Patients, the swabs from whose throats were found to contain the Klebs-Loeffler diphtheria bacillus, had their throats painted for three days with borax and glycerine, and their feeding-cups, spoons, etc., were for three days kept in separate basins of sterilising solution from those of the others, whose throats gave no result or only the Pseudo-Diphtheria bacillus. After the outbreak in December, 1900, however, the spoons, etc., were kept separate during the whole stay of the patient in hospital. (6) Toys used by any patient were to be kept solely for that patient, and put away in the locker belonging to the bed; no interchange of toys amongst the

children to be permitted. (7) No visiting to be permitted; or, if permitted, no toys or sweets to be brought by visitors. (8) Floors to be cleansed with a damp duster and not swept, so as to avoid stirring up dust. (9) No cats to be allowed in the wards. (10) All milk to be sterilised, and all water filtered through a Chamberland-Pasteur filter.

All these recommendations have been thoroughly carried out since the reopening of the hospital in October, 1899. Much praise is due to the matron, the sisters, and the nurses, for the great care displayed in the sterilisation above alluded to, which has involved a great deal of additional work and supervision. I am sure that these processes have been the most effective cause in producing the comparative freedom from diphtheria displayed by the hospital since the system was introduced.

The following Table shows the cases of diphtheria that have occurred in the hospital since the reopening on October 26th, 1899:—

Diphtheria Cases from October 26th, 1899, to Date.

Name.	Age and Sex.	Date of Admission.	Date of Attack.
A. K., Patient ...	F., 5	October 26, 1899	October 28, 1899
W. N., „ ...	F., 7	November 7, „	November 14, „
M., Nurse ...	F.		March 8, 1900
P. G., Patient ...	M., 8	September 1, 1900	September 9, 1900
L. S., „ ...	M., $\frac{10}{12}$	October 8, „	October 26, „
G. S., „ ...	M., 9	November 28, „	December 1, „
G. C., „ ...	M., 2	November 6, „	December 7, „
L. G., „ ...	F., 7	November 19, „	December 11, „
E. S., „ ...	F., 5	November 24, „	December 12, „
E. P., „ ...	M., 7	December 29, „	January 1, 1901
G. D., „ ...	M., 7	January 1, 1901	January 7, „
A. O., „ ...	M., 5	June 20, „	July 2 „
A. P., „ ...	M., 3	August 9, „	September 14, „
H. A., „ ...	M., $\frac{10}{12}$	September 12, „	December 4, „
S. B., „ ...	F., 8	October 31, „	November 26, „
J. H., „ ...	F., 3	November 14, „	November 29, „

There were no cases of diphtheria amongst in-patients or staff in 1902.

It will be seen that out of the above 16 cases only one was a nurse, all the others being patients. In 5 of the cases, less than seven days elapsed between admission and attack, so that possibly these cases were actually incubating the disease on admission. In 13 of these cases the throat was swabbed on admission. Three showed the presence of the K. L. Diphtheria Bacillus, two of these developing the disease within a week of admission; two

showed Pseudo-Diphtheria Bacilli, several weeks elapsing between admission and attack, and eight gave negative results from bacterioscopic examination. The four cases bracketed together formed the small outbreak in December, 1900, previously alluded to; after which the female ward was closed for fourteen days.

The total number of patients admitted to the hospital from October 26th, 1899, to the end of 1901, have been 1149. The number of swabs taken on admission was 814, or 62 per cent. of the patients admitted. Of the total 814, 88 or 10.8 per cent., showed the K. L. Diphtheria Bacillus; 162, or 20 per cent., showed the Pseudo-Diphtheria Bacillus; and 563, or 69.1 per cent., gave a negative result.

In 3 cases the Diphtheria Bacilli were tested as to their virulence. Two were found to be slightly virulent, and one very feebly, if at all.

—	No further Symptoms.	Dip. Bac. subsequently found in Swab.	Developed Nasal Discharge with Dip. Bac.	Developed Dip., and Removed to M. A. B.
88 cases admitted (A) with Dip. Bac.	84 (95 per cent.)	—	1	3 (3.4 per cent.)
162 cases admitted (B) with Pseudo-Dip. Bac.	159 (98 per cent.)	—	—	3 (1.9 per cent.)
563 cases admitted (C) with *nil*	549 (97 per cent.)	3	2	9 (1.6 per cent.)

The above Table shows that of the three classes of cases admitted, subdivided according to their having (A) K. L. Diphtheria Bacilli, (B) Pseudo-Diphtheria Bacilli, or (C) *nil* in their throat secretions, each gave nearly the same percentages of cases without further symptoms and of cases developing clinical diphtheria. The three cases which developed nasal discharge with diphtheria bacilli presented no other diphtheritic symptoms, and were treated in the ordinary way in the hospital. They were all infants under 1 year of age.

Very few of the cases were swabbed for bacterioscopic examination a second time; but a few were so treated, owing to suspicious symptoms or for other causes. Three cases, which were reported *nil* on admission, were thus on a second examination found to have K. L. Diphtheria

Bacilli present; but no clinical symptoms manifesting themselves, they were kept in hospital, and no untoward results followed.

Of the 3 cases which subsequently became affected in Class A, the attack of diphtheria developed three days, six days, and one month respectively, after admission. Of the 3 cases in Class B, in one case nasal diphtheria appeared three days after admission, but there is reason to believe that the child was actually suffering from the disease on admission. In the other 2 cases, twenty-five days and three months elapsed between admission and attack. In Class C the intervals between admission and attack were respectively three days, seven days, eight days, eleven days fourteen days, fifteen days, twenty-one days, twenty-two days, and thirty-five days.

The following notes are interesting, as showing the tendency of the K. L. Diphtheria Bacillus to persist in the throat secretions of certain children. A little girl of three years was taken into the hospital on March 14th, was discharged, and was readmitted on April 11th, and again on June 20th. On each admission the diphtheria bacilli were found, but no symptoms were ever present. Another girl of twelve was also twice admitted (September 11th and December 29th), and the diphtheria bacilli were present on both occasions.

When the hospital was reopened in October, 1899, the throats of all the staff (thirty-two in number) were bacterioscopically examined. In 29 cases the report was *nil*, in 2 cases Pseudo-Diphtheria Bacillus was present, and in 1 case K. L. Diphtheria Bacillus was present. This latter was the case of a nurse, aged 22, who was sent to the M. A. B. Hospital for diphtheria on August 20th, 1899. She was discharged on October 10th, and went to her home in the country for two weeks. She returned to work at the hospital on October 24th; but the result of the swab examination being known, she was isolated in the nursing home. On examination of her nose some ulceration was found, and antiseptic syringing was employed. A second swab taken on November 11th showed that the diphtheria bacillus was still present. She was then sent to the Convalescent Home at Broadstairs, and a third swab, after she had been there ten days, showed that the bacillus had disappeared. She returned to her duty at the hospital on November 25th. In this case, change of air at the seaside appeared to be more effective than antiseptic syringing.

The throats of twenty of the staff were also swabbed in

the middle of August, 1899, shortly after the hospital had been closed owing to the diphtheria prevalence. On this occasion two presented the K. L. Diphtheria Bacillus, four the pseudo-diphtheria bacillus, and fourteen were returned as *nil*, the percentage of diphtheria and pseudo-diphtheria swabs being much higher than in October, 1899, three months after the outbreak.

With the exception of the case of diphtheria in Nurse R., in March, 1900, the health of the staff has been very good since the reopening of the hospital in 1899. Three of the nurses have had scarlet fever in the past three years, and there have been 11 cases of tonsillitis or sore throat of a mild character, presenting no septic or diphtheritic symptoms, and in no case necessitating absence from duty for a longer period than eight days.

To sum up: in the two and a-half years, 1897—1898, to August 1899, 2,052 patients were admitted to the wards, and 65 cases of diphtheria occurred amongst them, giving a case-rate of 31 per 1,000 admissions. In addition, there were 9 cases of diphtheria and 16 septic throats amongst the staff of the hospital. From October 26th, 1899, to December 31st, 1902, 1748 patients have been admitted, and 15 cases of diphtheria have originated in the wards, giving a case-rate of 9 per 1,000 admissions. In this period there has been only one case of diphtheria amongst the staff, and no septic throats.

It seems to be a fair inference, from the facts above stated, that the diphtheria outbreaks of past years in this hospital have been due to the admission of children with diphtheria bacilli in their throat secretions, of low but potential virulence: that, owing to certain unknown conditions appertaining to hospital life, these bacilli have subsequently reacquired virulence, and have originated diphtheria, with its usual clinical manifestations, either in these self-same children, or in others to whom the bacilli had been transferred. Possibly, the somewhat overcrowded condition of the hospital, with fifty-six beds, was influential in modifying the virulence of the organisms, subsequent to the admission of the infected children. It is now generally recognised that one of the most potent factors in the transmission of diphtheria infection is direct contact, or transference of bacilli from the mouth of an infected child to the mouth or nose of a healthy child. The system of sterilisation of utensils introduced in October, 1899, aimed at the prevention of this transference of infection from patient to patient. On the whole, it appears to have

answered its purpose; for, with the exception of the small outbreak of diphtheria in December, 1900, when 1 in 80 carbolic was in use, all the other cases of this disease have been sporadic, and separated by long intervals of time. The system has certainly been effective in preventing infection of the staff, as shown by the facts above recorded.

Apart from the interest attaching to the history of this hospital in connection with other children's hospitals, institutions, and schools, where children are massed together, I think it has some bearing on the question of post-scarlatinal diphtheria in fever hospitals. The system of sterilisation of utensils aims at the isolation of the patient in his ward, so far as regards infections transmitted by direct contact. It is, of course, powerless against aerial infections. But we are all now tending to regard diphtheria and scarlet fever as diseases largely spread in the former manner. If the child sent to a fever hospital, whether with scarlet fever or diphtheria, can be isolated in his ward to the extent that he is not exposed to infection from his neighbours—either the infection of the disease from which he is not suffering (scarlet fever or diphtheria, as the case may be), or the more virulent form of infection of the disease from which he is suffering, which may very well happen under existing circumstances to be communicated to him—a great advance will have been made in securing true isolation in institutions which are now only isolation hospitals in a somewhat limited sense. The existing fever hospital is certainly "isolation" so far as the great body of the public is concerned, but is in no sense isolation of the sufferer amongst his fellow-sufferers.

The system of sterilisation advocated means extra nurses and more supervision; but I am of opinion that the results of its introduction into all hospitals, where children are received, would justify the extra expenditure incurred.

THE SEASONAL INCIDENCE OF TYPHOID FEVER AND OF DIARRHŒA.

THE SEASONAL CONSUMPTION OF SHELLFISH PROBABLY ONE OF THE CHIEF FACTORS AS REGARDS THE FORMER, AND THE SEASONAL PREVALENCE OF FLIES AS REGARDS THE LATTER.

By J. T. C. NASH, M.D.(Edin.), D.P.H.(Camb.), Medical Officer of Health, Southend-on-Sea.

Read: April 17th, 1903.

For some time past I have had prepared a short note on the Seasonal Incidence of Typhoid Fever, which I mentioned to Dr. Bulstrode, suggesting that it might be taken with some other short paper, at any convenient opportunity, if he considered it was likely to induce a discussion. When, however, about a week ago, Dr. Bulstrode asked me if I would read my paper to-night, owing to circumstances which unfortunately made it impossible for Professor McWeeney to read the paper he had promised, I felt it my duty to this Society to endeavour to elaborate my contribution a little more. Accordingly, I wrote to a few Medical Officers of Health, asking for figures showing the monthly Incidence of Typhoid Fever in their districts for a few years past; and I am much indebted to these gentlemen, whose names I shall presently mention in detail, for many of the facts I shall have the honour of laying before you.

My researches into the causation of Typhoid Fever in Southend, in 1902, embodied in a paper read before the Congress of the Sanitary Institute at Manchester, in September, 1902, and published in the *Journal of the Sanitary Institute*, vol. xxiii, Pt. iv, January, 1903, emboldened me to suggest for the consideration of epidemiologists that present ideas as to the causation of the Seasonal Incidence of Typhoid Fever are capable of expansion and modification. An explanation of the autumnal rise in the incidence of Typhoid Fever has been attempted by Pettenkofer's theory, as to the influence of the rise and fall of the subsoil water; though the best authorities in this country

have failed to trace any invariable relation between the onset of Typhoid Fever epidemics and the range of fluctuation of ground water. It is, however, granted that the theory reasonably explained the range of Typhoid Fever in some parts of Germany, where certain conditions as to the nature of the soil, level of the ground water, and possible sources of pollution, such as leaking cesspools, obtained.

Mr. Shirley Murphy has found distinct increase in the number of notifications of Typhoid Fever in London following on floods in the Thames Valley above the water intakes; but surely floods often occur—as often as at any other time of the year—in February, when the average incidence of Typhoid Fever in London is beginning to fall below the mean line. Dr. Bruce Low, on behalf of the Local Government Board, reported on Typhoid Fever in Southend in July, 1896, and quoted Ballard's hypothesis as to the relation between diarrhœal diseases and organically polluted soil, particularly when combined with the raising of the ground temperature to about 56 deg. Fahr. at four feet below the surface. It may be remembered that Tomkins, of Leicester, suggested a temperature of 60 deg. Fahr., at one foot below. I consider these circumstances of temperature as chiefly coincidental, or at most indirectly causative.

My chief object in reading this paper before the Epidemiological Society to-night, was to draw attention to the charts I now show, and the conclusion that I deduce from them, viz., that the seasonal incidence of Typhoid Fever in this country, particularly in well-drained towns with a good or properly filtered water-supply, has a closer relationship with the consumption of infected shellfish than with any meteorological or geological phenomena. It is, I take it for granted, the almost universal opinion, that, in this country, at any rate, Typhoid Fever epidemics are of water-borne origin. This is the case whether the source of contamination is the public water-supply, or the infection of milk, through the use of infected water for washing the cans, or by way of adulteration, etc.; and, obviously, it is still the case when shellfish are eaten which have been cultivated or laid in sewage-polluted waters. Sir Charles Cameron, of Dublin, twenty-two years ago, pointed out "the possible relation of Typhoid Fever to the consumption of specifically polluted oysters." Since Conn* directed atten-

* *Medical Record*, December 15th, 1894.

tion to oysters that had been "fattened" in waters polluted with typhoid evacuations as the practically conclusive cause of an outbreak of Typhoid Fever at the Wesleyan College, Connecticut, evidence has been yearly accumulating, inculpating shellfish from polluted waters as a causative agent of Typhoid Fever. The relationship of uncooked shellfish to Typhoid Fever was recognised by our esteemed colleague, Dr. Newsholme, Medical Officer of Health for Brighton, as long ago as March, 1894, when he laid before the Sanitary Committee of the Brighton Town Council the particulars of eight cases of Typhoid Fever, which had occurred during the preceding quarter, and which were attributed to oysters from polluted layings. The Brighton sanitary authority early took action, and the representations made to the Local Government Board were among the reasons which led to the appointment of Dr. Bulstrode to make a detailed inquiry into oyster culture in relation to disease. The result of that inquiry is embodied in Dr. Bulstrode's valuable *Report*,* which is familiar to all.

Dr. Newsholme's work in connection with this matter deserves special mention, because, on his initiative, a petition was promoted by Brighton, to which the sanitary authorities of twenty-nine great towns affixed their signatures, in the hope of inducing the Government to formulate legislation on the matter.

Sir William Broadbent, by a contribution to the *British Medical Journal*,† early in 1895 drew general medical attention to the causative agency of infected oysters as a source of Typhoid Fever. Numerous similar contributions have since been made; while scientific laboratory experiments have been made in large numbers by numerous observers. The part played by shellfish eaten raw has, therefore, been thoroughly recognised for some years, and I only refer to the matter to give due credit to the pioneers in this matter. In an inquiry into an outbreak of 23 cases at Shoeburyness in 1899, Drs. Thresh and Walter suspected cockles as the source of mischief, and adduced strong evidence in support of this contention.‡ During the past year I have been able to conclusively demonstrate that so-called "cooked" cockles may be a source of typhoid infection; and later experience in London and extensive laboratory experiments have confirmed my contention that

* *Report of the Medical Officer to the Local Government Board*, 1894 and 1895: Appendix.

† *British Medical Journal*, January 12th, 1895.

‡ *British Medical Journal*, December 16th, 1899.

cockles may contain, even *after* "cooking," the living virulent bacteria capable of inducing Typhoid Fever. The cooking of cockles as hitherto carried out is incomplete, and the interior of the cockle evidently does not reach a temperature as high as the thermal death-point of the typhoid bacillus. This is not to be wondered at, for the method of cooking is to merely plunge the cockles (which are placed in a sieve) for a few moments into boiling water until the shells open, when they are immediately removed from the water. Laboratory experiments have amply shown that this is insufficient to sterilise the interior fluids of the cockles. The same remarks apply to other shellfish from polluted sources, which hitherto have been overlooked as possible vehicles of infection, because they were supposed to be sterilised by cooking. Inquiries as to shellfish in the past should therefore have been extended to all forms of shellfish, whether "cooked" or raw when eaten. This being borne in mind as indicating an *under-estimation of shellfish typhoid in the past,* I now beg to draw attention once again to another factor of importance, and that is the "*close season,*" or indifferent season in this country for shellfish; for I contend that this probably has a material effect upon the seasonal incidence of Typhoid Fever. The "close" season for home and French oysters, as fixed by statute, extends from May 14th to August 4th. During the same period mussels and cockles are not in their prime, and are less eaten than at other times, though probably some shellfish are eaten all the year round. I beg to draw particular attention to, and make comparison between, the curves for Southend and London which I have here. The mean line for Southend represents a monthly average mean of five (notifications) for the years 1895 to 1901. It will be observed that, during the first seven months of the year, the monthly incidence is well below the monthly mean.

During August and September there occurs a tremendous increase in the notifications of typhoid fever, represented by a markedly precipitous rise in the chart curve. In the course of these two months the monthly incidence rapidly rises from 40 per cent. below the monthly mean in July, to 30 per cent. above in August, and no less than 190 per cent. above mean level in September. Now, it is during the latter end of July that the cockle season comes into full swing, reaching its height in August and September. From about the middle of August oysters are in season, and numbers of shellfish shops are open, while itinerant vendors also ply their trade. Coincidentally we find this gigantic

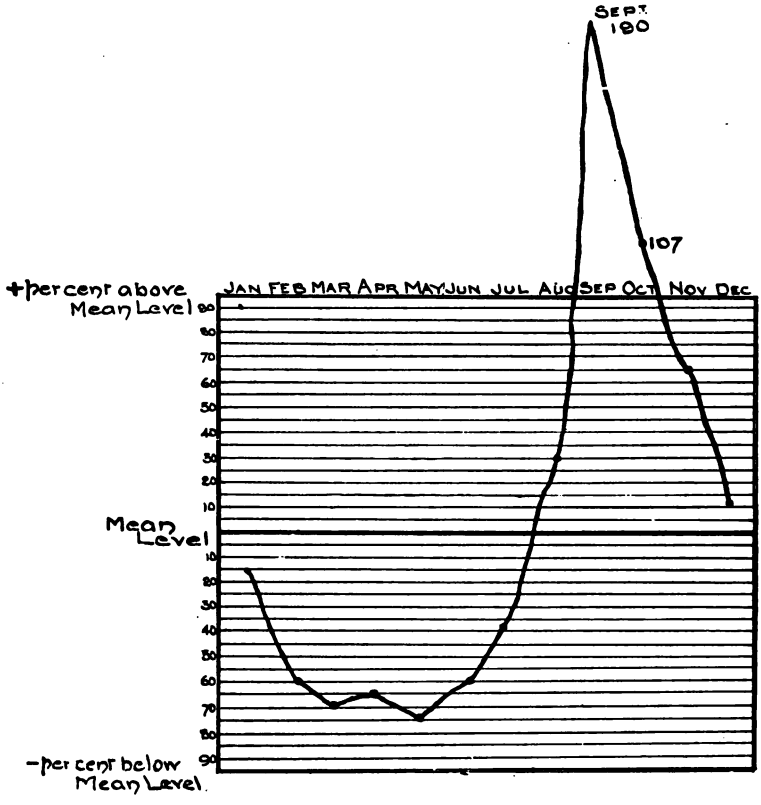


CHART I.—Southend, 1895 to 1901.

Southend-on-Sea.—Monthly Incidence of Typhoid for Years 1895 *to* 1901.

Years.	1895.	1896.	1897.	1898.	1899.	1900.	1901.	Total.
January ...	8	7	3	4	9	—	3	34
February ...	8	—	1	4	1	2	—	16
March ...	1	1	—	4	2	3	1	12
April	2	—	4	1	3	3	—	13
May	1	1	1	3	—	1	2	9
June	4	1	1	6	1	1	1	15
July	2	4	1	7	4	3	4	25
August ...	8	21	8	4	2	1	8	52
September ...	17	22	21	14	14	18	10	116
October ...	8	17	17	16	9	10	6	83
November ...	12	3	6	23	5	8	9	66
December ...	17	9	—	9	2	5	5	47

seasonal occurrence of Typhoid Fever. The seaside season begins to wane towards the end of September, and early in October it is over. Nearly all the shellfish shops close until the following year. Immediately there is a marked drop in the incidence of Typhoid Fever, until, in December, it is almost at the mean level. Compare this chart with that of London, which I have roughly imitated from the Registrar-General's Annual Summary for the year 1890, as reproduced in Stevenson and Murphy's great work on Hygiene. Here, again, during the close season for European oysters, and indifferent months for other shellfish, the typhoid curve is at its minimum. The holiday season comes on. Those who are away during July return in August, when the curve begins to ascend. I am tolerably certain that, if severe cross-examination were made, it would be found that a large proportion of the cases had

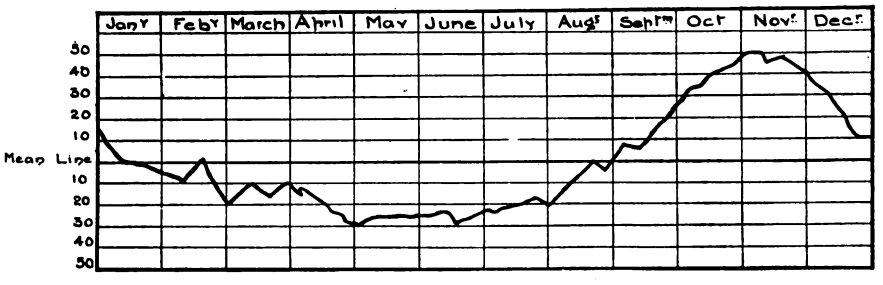


CHART II.—London.

indulged in shellfish while away at the seaside. During August—the great holiday month—the Londoner is enjoying himself to the full at the seaside; then he returns to London, and immediately the typhoid curve rises higher in September; but it has not yet reached its zenith. Whereas at the seaside resort the local shellfish trade languishes and almost expires in October, it is now transferred to London. October and November are the great months for dinners, and the festive oyster is in much evidence and great demand. It is at this time we find the typhoid curve at its height in London. Truly, a noteworthy coincidence. The single coincidence, as displayed by one chart alone, is suggestive; but the double coincidence, on a careful comparison of the two charts, is more than suggestive, and indicates a line of inquiry which it seemed to me should lead to some definite results. Among cases attributable to infected shellfish should be included cases arising from

contact with shellfish cases, and those cases which arise from the handling of infected shellfish, even if they are not eaten. Both Dr. Newsholme and I have previously drawn attention to this last important point. I now show a curve I have worked out for Edinburgh, from figures supplied me by the courtesy of Sir Henry Littlejohn, Medical Officer of Health for that city.* The chart is more irregular than those for London and Southend, but resembles that of the latter in the very pronounced epidemic peak in September. The monthly notifications are above the average monthly mean line of 23.9 cases from May to October, and below from

* The curve chart has, unfortunately, been lost; but the figures for Edinburgh, from which it has been constructed, are as follows :—

City of Edinburgh.—Monthly Notifications of Typhoid Fever during the undermentioned Years.

Year.	January.	February.	March.	April.	May.	June.
1894	11	21	7	17	37	29
1895	29	21	26	16	22	24
1896	31	29	18	42	39	21
1897	13	7	12	30	25	21
1898	15	13	16	21	22	27
1899	9	5	13	13	28	28
1900	15	9	16	21	24	28
1901	16	11	11	7	14	12
Totals ...	139	116	119	167	211	190
Monthly Means for Years 1894 to 1901 ...	17.3	14.5	14.8	20.8	26.3	23.7

Year.	July.	August.	Sept.	October.	Nov.	Dec.	Total.
1894	40	33	33	24	28	30	310
1895	46	35	74	52	38	34	417
1896	36	34	21	30	10	17	328
1897	22	18	31	43	21	11	254
1898	13	16	35	27	18	18	241
1899	42	37	43	31	24	16	289
1900	27	25	32	17	21	14	249
1901	19	26	34	29	24	12	215
Totals ...	245	224	303	253	184	152	2303
Monthly Means for Years 1894 to 1901 ...	30.6	28	37.9	31.6	23	19	—

Average Monthly Mean ... ... 23.9.

November to April, with a marked tendency to rise in April. The perturbations in the Edinburgh curve, I think, indicate a variety of causes at work; but I shall not be surprised to learn that the marked peak in September is coincidental with an enormously increased consumption of shellfish during August and September. I hope Sir Henry Littlejohn will be able to enlighten us on this matter.

By the courtesy of Dr. Elliston, Medical Officer of Health for Ipswich, I am able to give the monthly notifications of Typhoid Fever in Ipswich for 1902.

Ipswich, 1902.

Week ending—	
January 26th	3
February 28th	5
March 30th	3
April 27th	5
May 25th	7
June 29th	17
July 27th	39
August 31st	25
September 28th	37
October 26th	16
November 30th	14
December 28th	2
Total for 12 months ... ... ∴	173

It will be observed that the main incidence of Typhoid Fever was during the usual shellfish season. Dr. Elliston himself is distinctly of opinion that about 25 per cent. of these cases were due to shellfish, chiefly cockles, taken from the Orwell, a sewage-polluted river, which at low tide is reduced to a comparatively small volume, and is then highly polluted. My own opinion is that closer investigations and severer cross-examinations as to the handling of shellfish, or as to contact with previous shellfish cases, would have shown an even larger proportion as directly or indirectly connected with shellfish.* Dr. Elliston could only supply me with the yearly instead of the monthly totals for previous years. From these, however, I have constructed a diagram,† which I think justifies an assumption that contaminated food rather than contaminated soil must have played the principal part in epidemic years. Dr. Elliston himself remarks: "It has become increasingly clear that we have in polluted shellfish an explanation of many of the sporadic outbreaks of Typhoid Fever in

* For various reasons, persons often deny eating shellfish when first questioned, though admitting it later.

† Not reproduced.

Ipswich during the last three summers, the causation of which has been mysterious" (*Annual Report*, 1902, p. 23).

Dr. Russell, of Great Yarmouth, has kindly favoured me with figures showing the monthly incidence of Typhoid Fever in his district. If these figures be carefully examined and a chart (Chart III) constructed, it will be found that the seasonal rise and fall of Typhoid Fever is in fairly close correspondence with the maximum and minimum consumption of shellfish. Unusual numbers of cases in the winter seasons of 1898 and 1899 have raised unduly the average readings for January, February, and March, in the curve for the five years 1897-1902. These cases were attributed by Dr. Russell to the consumption of mussels taken from the Yare. The sale of these was stopped in 1900, with a con-

Great Yarmouth.—Monthly Incidence of Typhoid for Years 1897 *to* 1902.

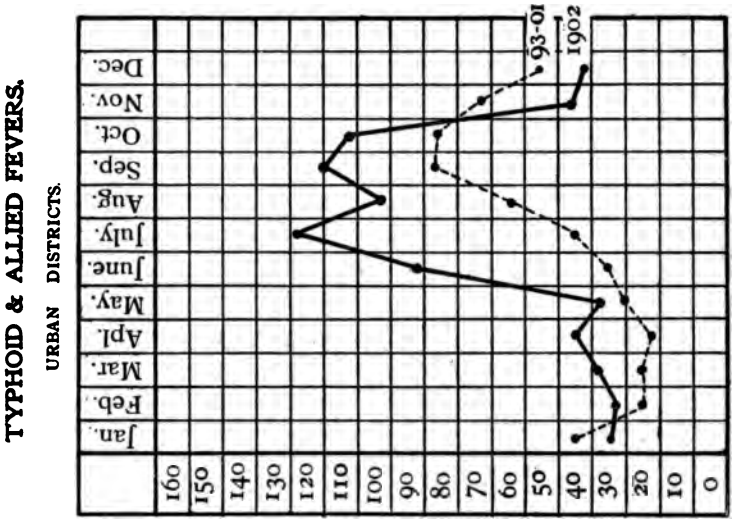
Years.	1897.	1898.	1899.	1900.	1901.	1902.	Total.
January	16*	25*	40*	5	3	3	92
February	17	38	25	5	5	5	95
March	11	32	3	2	15	3	66
April	2	17	3	2	20	1	45
May	7	16	3	5	5	1	37
June	3	7	4	3	4	0	21
July	5	8	2	2	4	4	25
August	15	9	6	9	3	6	48
September	10	13	25	10	10	7	75
October	15	26	7	52	12	3	115
November	15	28	4	23	4	3	77
December	15	25	10	5	2	2	59

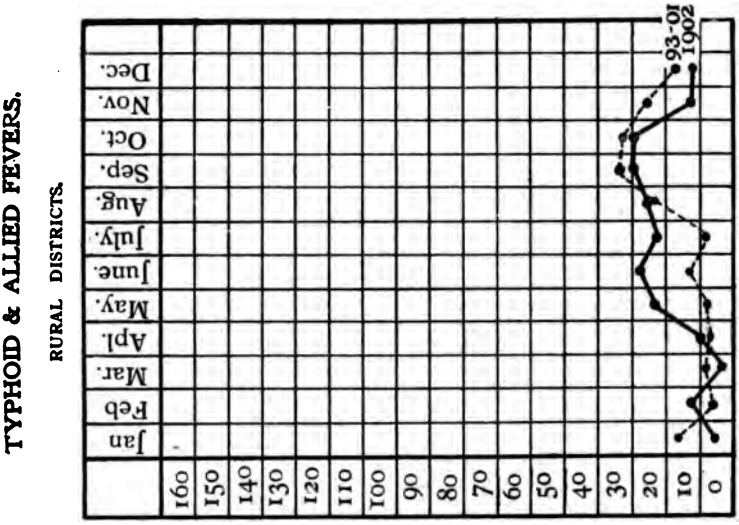
sequent marked diminution in the number of cases notified. I have indicated, by a second dotted curve, the incidence of Typhoid Fever in Yarmouth during 1902. The wisdom of having stopped the sale of mussels from the Yare is evident. Though there is still a slight autumnal rise, yet the highest reading in September is actually 30 per cent. below the average monthly mean for the five preceding years: a very remarkable and noteworthy reduction.

Dr. Tubb Thomas, Medical Officer of Health for the county of Wilts, in reply to a query of mine, says "the majority of the cases of sporadic typhoid occurring in the county are very obscure in their origin, but with the greater number there is evidence of having partaken of

* Excess of typhoid fever in the winter months of these three years attributed to mussels taken from the Yare.

CHART IV.





shellfish during the period of incubation, and immediately before."

This is interesting in view of Wiltshire being an inland county.

Dr. Thresh, Medical Officer of Health for the county of Essex, in a Summary of the Reports of the District Medical Officers of Health for the year 1901, says (p. 52): "Typhoid Fever is about three times more prevalent in the districts bordering on the Thames than in the rest of the county; and in this area of excessive prevalence the greatest excess is constantly in the districts at the mouth of the river."

I have suggested that the reason for this excess lies in the fact that shellfish are naturally more freely consumed in this "area of excessive prevalence" ("Special Report on Typhoid Fever in Southend, 1902").

I find that in his Summary for 1902 Dr. Thresh has published charts which are of the greatest interest. They illustrate the incidence of "Typhoid and Allied Fevers" in the Urban Districts and in the Rural Districts of Essex, with total populations of 593,577 and 240,118 respectively. I have ventured, with the kind permission of Dr. Thresh, to reproduce these charts on the preceding page. They show the monthly incidence not only for 1902, but the monthly mean for the years 1893-1901. They resemble the Southend Typhoid Curve, in that the incidence of Typhoid Fever is abruptly precipitous in August and September; but the peak is more rounded, though the fall in November and December is fairly rapid, in marked contrast to the behaviour of Typhoid Fever in London, which, as we have seen, does not reach its height till November, and then gradually falls, not, however, reaching the mean line until February, and only thereafter falling below it.*

From the Annual Report for 1901 of the Medical Officer of Health for Morecambe, a small seaside town of about 12,000 population, I find that out of thirteen notifications during the year no less than ten occurred during August, September, and October. No note is made as to the causal agent, but, as with most other seaside towns, a system of sewerage with outlets into the sea is still in vogue.

Of significant import is Dr. Niven's Report on Typhoid Fever in Manchester during 1901. Here, again, the minimum monthly number of notifications occurred in May, while the maximum occurred during August, September, and October. The monthly number of notifications was below a mean level of thirty notifications a month, from December to July; while during August, September, and

* The curve for 1902 is interesting. The abnormal incidence in June and July, and the generally higher curve through the shellfish months, is noteworthy as coincidental with a very high incidence of typhoid at Leigh, the centre of the cockle industry in Essex.

October, it was from 130 per cent. to 133 per cent. above mean level, falling in November to only about 33 per cent. above, and in December to 50 per cent. below. By means of an elaborate Table and diagrams, Dr. Niven endeavours to show a general correspondence between the dates of onset of the autumnal cases and a heavy rainfall, described as the "critical rainfall," occurring from three to five weeks before; necessary concomitant features in his opinion, being a preceding or subsequent period of dry—or comparatively dry—weather, and an ascending ground temperature. I must confess that I am by no means convinced that Dr. Niven fully makes out his case—even from a mere coincidental point of view, let alone a causal. Then, again, these conditions as to rainfall, drought, and temperature are *general* conditions, affecting more or less the

Manchester, 1901.

Week ending—	
January 26th	13
February 28th	12
March 30th	18
April 27th	18
May 25th	9
June 29th	16
July 27th	13
August 31st	66
September 28th	70
October 26th	70
November 30th	40
December 28th	14
Total for 12 months	359

Monthly mean, about 30 notifications a month.

whole population of a city or other district. In the case of Manchester these conditions would be common to a population of about 550,000 persons, and yet, during each month of the greatest prevalence of Typhoid Fever (August, September, and October) only about 1 person out of every 10,000 inhabitants suffered from Typhoid Fever in each whole month, or only about 1 person in every 3,000 during the whole three months. Surely, if such a general coincidence were directly causal, we would have a larger proportion of persons attacked. From this small proportion, too, must be subtracted those cases during the autumn which are definitely, or with great probability, traced to a definite cause: such as secondary cases; cases due to shellfish; and cases probably due to a possibly infected milk supply, etc.

Dr. Niven states that during the year there were twenty-five cases, "with a history, more or less definite, of having

eaten shellfish within a period of from one to three weeks before the onset of illness." In my opinion this probably does not exhaust the real number of cases due to shellfish: such, for instance, as those with a very short or a protracted incubation period; those who acquired infection by the *handling* of infected shellfish; and those who unknowingly had been in contact with the person or excretions of a a former shellfish case. Be this as it may, it is clear from Dr. Niven's Report that 22 of the 25 cases attributed to shellfish occurred during the second half of the year (July to December), as compared with only 3 cases during the first half of the year.

I believe that in Brighton Dr. Newsholme attributes about 30 per cent. of the cases of Typhoid Fever, directly or indirectly, to shellfish. In my own district I consider shellfish to be connected with at least 70 per cent. of the cases which occur.

An important but not easy point to determine is what proportion of a population does the shellfish-consuming section bear to the section which does not eat shellfish? During 1902 I had inquiries made at 656 houses in connection with notified cases of infectious disease (including chicken-pox). These inquiries were made in all parts of the Borough. As a result I found that, with only two exceptions, the only shellfish eaters in this inquiry were found in houses in which Typhoid Fever had occurred. Another important fact is that nearly all the local shellfish shops close in October, because there is so little local trade.

In a Special Report I worked out the ratios for the four months, June to September, 1902. The standard terms of the ratios in one year per 1,000 persons living in each section of the population (calculating the shellfish-eating section at 5 per cent. of the total, a proportion probably greatly in excess of the real number) were calculated to be as follows:—

For the entire population the attack-rate was	5.0	per 1,000.
" shellfish-eating section .	56.0	"
" shellfish merchants and employées . . .	160.0	"
For the remainder of the population, even including those secondary to shellfish cases, only , . . .	2.3	"

I have constructed a diagram, which I show here, to illustrate the disproportion between 5 per cent. and 95 per cent. of the population, and the number of cases of Typhoid

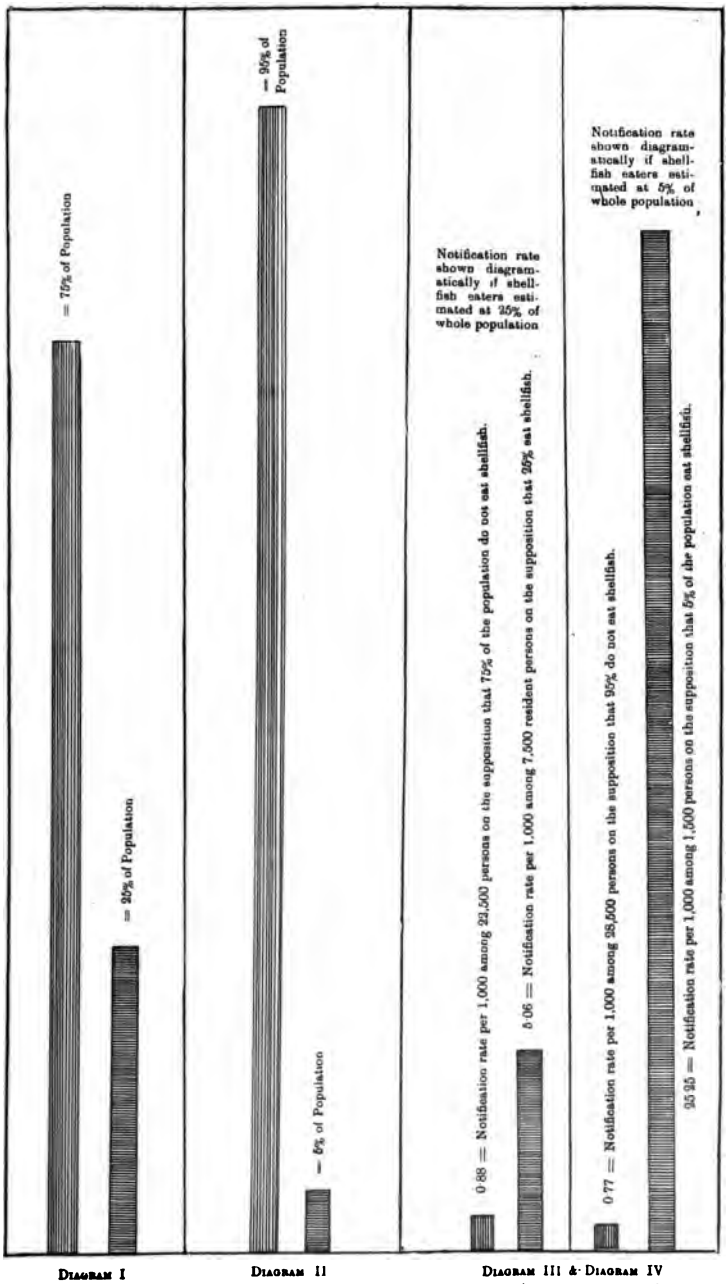


DIAGRAM I
Represents the disproportion between the two sections of the community if 25% eat shellfish

DIAGRAM II
Represents the difference if 5% eat shellfish

DIAGRAM III & DIAGRAM IV
Show the enormous preponderance of typhoid fever among shellfish eaters, whether they be estimated at 5% or even 25% of the total population

CHART V.—Southend. Diagrams showing Incidence of Typhoid Fever on *those who eat* Shellfish, as compared with those who abstain from Shellfish.

Fever occurring in each section. I am certain that the shellfish-eating section is less than 5 per cent. of the whole population; but, assuming this is an approximate proportion, the incidence of Typhoid Fever on the shellfish-eating section was at least thirty-six times as great as on the non-shellfish-eating section—*cæteris paribus.*

I now propose to deal with the figures for Brighton. They were kindly sent me two days ago by my friend, Dr. Newsholme. I have constructed a chart from these figures, but before I saw them I ventured to write my opinion as follows:—The monthly incidence of Typhoid Fever in Brighton will probably differ from that of London, and that of Southend, and most other seaside resorts, for the following reasons. Brighton has a definite winter season as well as an ordinary summer season. During the summer season probably all sorts of shellfish are eaten, more particularly mussels, which are readily obtained from a neighbouring district. During the winter season the Brighton visitors are largely of the well-to-do class, who spend some time in Brighton, and can and do indulge in oysters. Therefore, Brighton probably has a considerable winter shellfish consumption, only somewhat less than the summer season consumption, and, therefore, I expect to find a heavier comparative winter incidence of Typhoid Fever in Brighton than in Southend, or other seaside towns which enjoy only a summer season. All the same, I shall expect to find the incidence of Typhoid Fever at its minimum during May and June. In other words, the Brighton curve probably will more closely resemble the London curve than the curves of other seaside towns.

Brighton.—Monthly Incidence of Typhoid for Years 1898 *to* 1902.

Years.	1897.	1898.	1899.	1900.	1901.	1902.	Total.
January	—	8	15	5	7	7	42
February	—	12	7	5	2	3	29
March	—	6	7	1	5	2	21
April	—	6	9	4	1	3	23
May	—	4	14	8	2	3	31
June	—	8	18	3	3	3	35
July	—	10	24	3	1	7	45
August	—	12	19	8	2	5	46
September	—	24	24	10	3	5	66
October	—	17	23	7	6	8	61
November	—	6	13	13	9	8	49
December	—	17	9	16	5	11	58

The chart I now show, which I have constructed from the figures kindly sent me by Dr. Newsholme, justifies the prediction I ventured to write before I knew the actual figures (see Chart III).

In the next place, I am indebted to Dr. Hope for the monthly notifications in Liverpool for the last seven years. From these I have constructed a chart which shows the usual autumnal incidence of Typhoid Fever (see Chart III). If shellfish play a prominent part in the causation of Typhoid at Liverpool, this curve appears to indicate a much smaller consumption in the winter months than in London. I trust Dr. Hope will be able to state whether this is so or not.

Liverpool.—Monthly Incidence of Typhoid for Years 1895 *to* 1902.

Years.	1895.	1896.	1897.	1898.	1899.	1900.	1901.	1902.	Total.
January ...	94	50	47	51	70	67	48	100	527
February .	64	44	35	40	59	28	41	79	390
March ..	62	50	41	52	70	44	43	57	419
April ...	94	68	52	66	81	42	26	63	492
May ...	186	88	47	60	58	64	52	83	638
June ...	101	95	47	41	89	36	39	63	511
July ...	99	111	67	48	69	50	31	83	558
August ...	104	141	118	79	68	54	104	82	750
September	136	108	132	104	122	100	141	113	956
October ...	145	128	179	111	124	111	150	124	1,072
November	153	116	143	117	93	72	110	113	917
December.	68	64	83	94	85	63	79	66	602

In the instructive, interesting, exhaustive, and erudite Milroy Lectures which Dr. Corfield delivered last year, he drew attention to the fact that "Whereas the seasonal prevalence of Typhoid Fever is normally in the autumn months, this disease is more prevalent and fatal in London during the winter months. He stated that since 1890 he had pointed this out for St. George's, Hanover Square, and he quoted a Table from his Report for 1900, to show that during every year, except 1897, the monthly average for November and December was higher than that for August, September, and October. He pointed out that this rise in the number of cases of Typhoid Fever reported in the months of November and December was 'coincident with the increase of organic matter in the drinking water due to the flooded state of the river.'" He fully quoted the differences of opinion between himself on the one hand, and Prof. Sir Edward Frankland, Sir William Crookes, and Prof. Dewar, on the part of the Grand Junction and Chelsea

Water Companies, on the other hand, arising out of the temperate statements in his Report that: "It may be something more than a coincidence that the exceptional prevalence of enteric fever in London during November, December, and January, which are not the months for the seasonal prevalence of this disease, occurs in the very months in which London is supplied with water which contains an excess of organic matter."

He quoted some significant facts from Mr. Shirley Murphy's *Report* for 1894, and reiterated his opinion that "the unseasonable prevalence of Typhoid Fever in November and December in London is due to the distribution of inefficiently-filtered river water containing the poison of the disease, in sufficient amount, however, only to attack the most susceptible persons among the population."

With all due deference, I beg to submit that the coincidence as to the consumption of shellfish in London, to which I have had the honour of drawing particular attention to-night, is, at any rate, worth due consideration, and in my humble opinion is more consistent with the *comparatively* small increase in the number of cases of Typhoid Fever in London in November and December, than the pollution of a common water-supply.

I am bound to confess, that however slight might be the *specific* pollution of the water-supply of a great community, I should expect to find a larger proportion of cases than the comparatively slight monthly excess shown in London in November and December, as compared with September and October.

Professor Corfield has most conclusively proved that typhoid epidemics are almost invariably water-borne; and I beg to submit, that in the consumption of infected shellfish we are, as with milk, still really dealing with a water-borne infection.

I do not presume to contend that his assumption as to the relation between Typhoid Fever and an increase of the organic matter in the water in winter is a mistaken one.

On the contrary, I am disposed to think that in the bodies of a few particular individuals this condition of the water may form a necessary link in a chain of evolutionary conditions, by means of which specific pathogenic properties may be acquired by one of the members of the *Coli* group of organisms, which so closely resemble *B. Typhi Abd.* If, on the other hand, the specific organism of Typhoid

Fever is actually present in the water, *B. Coli* ought, I think, to be also present in sufficient numbers to be detected by ordinary methods of bacterioscopic examination. If *B. Coli* is not found in unusual numbers, I should consider the evidence as distinctly against specific pollution, in spite of the increase in organic matter in the water.

Apart from these considerations, I merely suggest that the increased consumption of shellfish in London in the winter months is an alternative explanation which perhaps will meet the facts more convincingly than the small increase of organic matter in the water.

I am strongly of opinion that the autumnal incidence of Typhoid Fever is largely due to the increased consumption of shellfish at that time of year; it is also partly due, no doubt, to the eating, in larger quantities than usual, of doubtful ice-creams, and of salads and other uncooked vegetables, which have been subjected to manurial pollution.

Typhoid fever being a communicable disease, it follows that the greater the number of primary cases which occur at a given season—the more secondary cases which necessarily are likely to follow. This fact must not be lost sight of in accounting for seasonal incidence. In my opinion, secondary infection plays a considerable part in the autumnal incidence of Typhoid Fever, whether by means of personal infection, or by the agency of flies, etc. These secondary cases should be referred back to the original causes of the primary cases, on which they are really dependent. If this were done, the percentage of cases traceable to shellfish would be found to be considerably augmented.

It is astonishing and almost incredible that, only so recently as 1900, it should have been necessary for this Society to hold a discussion on the infectivity of Typhoid Fever, and for Dr. Goodall to have to point out that some of the leading text-books taught that there was no risk in nursing Typhoid Fever patients, and no necessity for removing a typhoid patient to hospital.

It really seems past belief that anyone who has had to deal with the administration of a fever hospital, or with any considerable number of private typhoid patients, could have escaped both the experience and conviction that the disease was infective in the absence of intelligent nursing and efficient disinfection (the latter, of course, including not only the excreta, but also the hands of the nurse after

every occasion of attending to the wants of the patient, even to apparently so small a matter as smoothing the sheets, etc.)

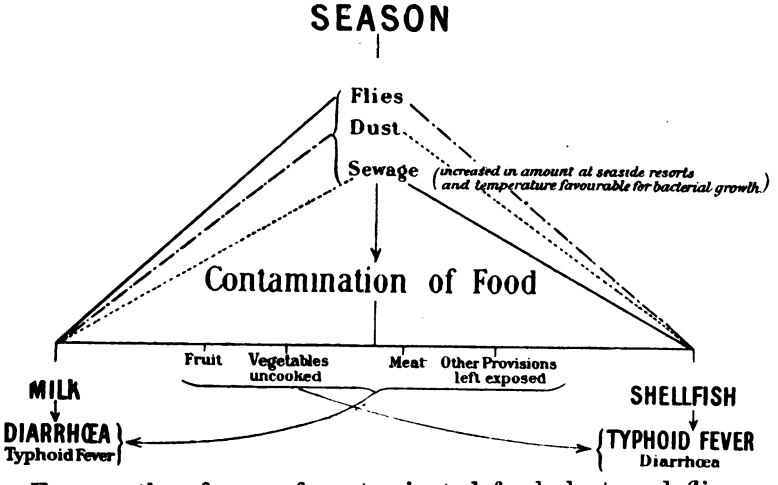
The part played by flies in carrying infective material during the summer months, is, in my opinion, not inconsiderable. My experience at Southend, in connection with diarrhœa, bears this out. I alluded to this matter in the part I took in the discussion on Professor Délépine's paper, read here in December last, on "The Bearing of Outbreaks of Food-Poisoning upon the Etiology of Summer Diarrhœa." From an investigation as to the incidence of diarrhœa in Southend during 1901 and 1902, and as to the coincidental prevalence of flies, it is apparent that, whereas there occurred in Southend during July and August, 1901, no fewer than 23 deaths from diarrhœa among infants under one year of age, there was *no* fatality from this cause during the corresponding months (the usual diarrhœa months) in 1902. Since June, the 4-ft. earth-temperature had exceeded 56 deg. Fahr. (Ballard's critical temperature), and yet there was no diarrhœa. As to rain, the weather was showery, but not extremely wet. Rain fell on twenty-two out of the sixty-two days in July and August. The conditions, therefore, should have been favourable for microbic growth, if, as suggested by Ballard, summer diarrhœa is probably dependent on some form of microbe in the soil which requires these essential conditions as to temperature, etc., for its development. My own suggestion is, that the usual seasonal circumstances are essential for the multiplication of the common house-fly and bluebottle fly.

The most remarkable phenomenon, to my mind, during June, July, and August, 1902, was *the almost complete absence of flies*, so much so that it was a matter of common comment, "There seem to be no flies this summer." In September, 1902, flies were noticed in Southend for about three weeks, and coincidentally 13 infant deaths from diarrhœa were recorded. Then the fly suddenly disappeared again, and with it also disappeared the sudden outbreak of diarrhœa.

The coincidence is a striking one. When I state that flies taken from refuse-heaps have been shown by Mr. Foulerton to harbour such organisms as *Bacillus Coli*, *Proteus Vulgaris*, and Gärtner's *Bacillus Enteriditis* (among others), it will be readily admitted that they might, indeed, play a very important part in the seasonal incidence of diarrhœa; and, if they happen to meet with unsterilised

crude typhoid excreta, of Typhoid Fever also.* I have ventured to draw a simple scheme to show the relation between season, flies, and sewage and dust-contaminated food, and diarrhœa and Typhoid Fever, putting these into larger and smaller type, according to what are, in my opinion, the more important factors in this connection.

Food, especially milk, contaminated by flies is probably the chief causal factor in summer diarrhœa; while, probably only a minor contributory cause to the ordinary incidence of Typhoid Fever. (Of course, I exclude epidemics due to grossly polluted milk.) On the other hand, I give the chief place to shellfish, polluted by sewage, as the principal causal factor in the autumnal rise of Typhoid



Fever; other forms of contaminated food, dust, and flies, being, in my opinion, merely contributory factors.

As Dr. Corfield pointed out in his Milroy Lectures, Dr. Bulstrode has shown, by most interesting maps and charts, "that whilst enteric fever has been undergoing enormous diminution in this country, the areas of both its maximum and minimum incidence have remained practically the same during the twenty years—1871 to 1890—which have witnessed such great improvements in the sanitary circumstances of our towns and villages" (*Twenty-Sixth Report of the Medical Officer of the Local Government Board,* 1896 *and* 1897).

* Experiments by Firth and Horrocks have proved that ordinary house flies (*Musca domestica*), can convey infective matter from specific excreta or other polluted material to objects on which they may walk, rest, or feed. (*British Medical Journal*, September 27th, 1902).

These sanitary circumstances (which include pure water-supplies, the keeping pure of the soil under houses, and the air in and around them, by satisfactory methods of removal of excreta and refuse, such as the provision of water-tight house-drains; their disconnection from sewers, cesspools, etc.); have, indeed, effected a marked reduction in the average annual death-rate from continued fevers (including Typhoid), and diarrhœa throughout the country; but Professor Corfield's Tables V and VI appear to indicate that we have almost reached the limit of the salutary effect of these sanitary improvements, and that there is still *a residuum of Typhoid Fever and diarrhœa due to other causes.* Dr. Bulstrode's charts and remarks as to the areas of maximum and minimum incidence are of special interest in this connection.

I submit that, just as—in the words of Sir John Simon, quoted by Professor Corfield—"it ought to be an absolute condition for a public water-supply that it should be incontaminable by drainage"—so, too, it ought to be an absolute condition that other foods for public consumption, such as shellfish and milk, should be incontaminable by drainage, whether this drainage be crude sewage, or a sewage effluent which has been merely treated by contact beds, without the land treatment so rightly advocated by the Local Government Board. Again, not only should our dairies, cowsheds, and milkshops, and other provision shops be administered on sanitary lines, but special attention should be given to the proper disposal of town refuse, and the *covering over* of all foods, whether cooked or not, so as to prevent the access of flies. Flies, when numerous, are such pests, that people are not slow to avail themselves of fly-catchers to reduce their numbers; but it is a question whether we should not more minutely study the natural history of flies, so as to destroy them in their larval forms, in the same manner as we wage war against the mosquito for the prevention of malaria.

As far as diarrhœa is concerned, I think the prevention of the contamination of foods (particularly milk) by dust and flies will effect a notable reduction in the incidence of this disease; but, as far as Typhoid Fever is concerned, I agree with Sir William Broadbent that, "whatever importance might attach to flies and dust, they are subsidiary to the infection by water"—in this country, at any rate, and probably in tropical countries also.

On the other hand, as regards shellfish, I think, if we could prohibit the sale of such as come from any layings

open to the slightest suspicion of sewage-pollution, unless prepared by cooking in such a manner that every part of them shall be exposed to a temperature of at least 70 deg. Cent. for at least fifteen minutes, we should, in my opinion, solve the greater part of the problem of the residual typhoid which appears to be unaffected by the present achievements of sanitary science.

I might, perhaps, be allowed to make a remark on the Influence of Sex and Age.

Influence of Sex and Age.—According to the Registrar-General, whose conclusions are based on a study of the admissions to the Metropolitan Asylums' Board Hospitals and the London Fever Hospitals, it would seem that more males than females are attacked between 5 and 20 years of age. I suggest that a partial explanation is probably to be found in the fact that rather more males than females partake of shellfish between these ages, and that larger quantities of shellfish are eaten by a larger proportion of young persons than by persons at ages below 5 or above 25 years of age.

I ascertain from shellfish merchants that the majority of their customers are young persons—as might indeed have been expected.

Having brought forward so much evidence in favour of the view that the seasonal incidence of Typhoid Fever in the late summer and autumn months in the country generally, and the early winter months in London particularly, is probably in large measure due chiefly to the fact that this is the chief period of shellfish consumption, I need only refer you to my Annual Report for 1902 for further details of local—and I hope also—general interest.

Our sanitary authorities generally pay proper attention to water-tight house drains, disconnected from the sewers; and provide admirable means for the *collection and removal* of house refuse. It is my opinion that if proper measures are also taken for the effective and speedy *disposal* of refuse and street sweepings, so as to limit the excessive breeding of flies, and the chances of flies carrying hurtful germs to food (the natural history of the fly being also closely studied, so as to devise means of controlling its general existence), and, failing this, if foods of all kind are properly protected from flies, I think we shall see a great reduction in the incidence of diarrhœa, and a moderate reduction in the incidence of Typhoid Fever.

A proper control of shellfish will, I think, secure a notable reduction in the seasonal incidence of Typhoid Fever, and

a moderate reduction in the incidence of diarrhœa. Both sets of measures together will, I think, considerably reduce the incidence of both forms of disease.

At any rate, these premises and conclusions appear to me to form something more practical to work upon than the ingenious theories built upon undoubted coincidences, but which I have tried to show, are probably only *indirectly* causal.

As far as results go in my own district, by drawing special attention to shellfish as a cause of Typhoid Fever, and thereby securing less consumption on the one hand, and better methods of laying and cooking on the other, and greater care in the selection of shellfish from purer layings, I may say that three months have now elapsed since I last received a notification of Typhoid Fever. This may be merely a coincidence, but I venture to think otherwise.*

* *Note made September 23rd.*—Since constant publicity has been given to the dangers of shellfish during the last nine or ten months, there has been a very notable reduction of typhoid fever in London, Southend, and elsewhere. In Southend, up to date (September 23rd, 1903), there has been a very notable reduction of cases originating in the Borough, as compared with the corresponding period of any previous year or the average figures for the years 1895-1902, the figures for which are available for comparison.

The figures for the years 1895 to 1903 (January to September), with their estimated population, are given below :—

1895.	1896.	1897.	1898.	1899.	1900.	1901.	1902.	1903.
51	57	40	47	36	32	29	76	18
16,203	17,609	19,302	22,583	24,710	27,721	29,479	31,892	34,940

Among the 18 cases notified to September 23rd, 1903, in no less than 17 has a history of shellfish been traced. The eighteenth case was apparently traceable to watercress.

Further note made November 5th, 1903. An interesting sequel is as follows :—During the latter end of August and September, a large number of labourers were imported into Southend, in connection with the building of a new sea wall. The majority of these men had not heard of the dangers attaching to shellfish, and, finding a plentiful supply of oysters to hand a little way out, where the Leigh Swatch enters the main waters of the river Thames, several partook freely of them. The result was a considerable incidence of typhoid fever and diarrhœa among these men, especially during the last week of September and the first week in October. When I ascertained that these labourers were partaking freely of shellfish, I personally visited each gang, warning them of the dangers, and informing them that several of their mates were stricken with illness. Printed notices were also placed along the foreshore, along the line of works, warning the public not to eat shellfish picked up in this situation. These notices were put up on September 30th, and nearly every man on the works forthwith ceased to eat oysters, etc. Since October 9th, no further cases of typhoid fever have occurred among these labourers, though further cases have occurred among other persons who have partaken of shellfish bought from hawkers in the streets.

I sincerely trust I have not seemed quixotic or needlessly iconoclastic in my interpretation of the views of eminent sanitarians of tried ability and great experience, whose views, indeed, I treat with great respect, and which are, perhaps, more entitled to your consideration than my own, and whose good opinion I value most highly.

I have merely tried to state and justify my own views and opinions, suggesting that they may better meet the explanation of the seasonal incidence of Typhoid Fever and diarrhœa than those hitherto held. These latter, I wish distinctly to acknowledge, have, in my opinion, undoubted influence on the seasonal incidence of these diseases; only I consider such influence more *indirect* than the explanation I have ventured to offer for the serious consideration of the Society to-night.

It only remains for me now to thank the Society for a patient hearing, and to invite discussion; at the same time craving your indulgence on the score that the greater part of this Paper has been rather hurriedly written within the last two or three days.

Discussion.

On the termination of Dr. Nash's paper an interesting discussion ensued, in which the following members took part:—Dr. Franklin Parsons, Dr. Newsholme, Colonel Lane Notter, Dr. Goodall, Dr. Louis Parkes, Dr. Butler, Colonel Davies, R.A.M.C., Mr. Shirley Murphy, and Dr. Bulstrode. Some of these gentlemen have been good enough to comply with the request of the Chairman that they should forward an abstract of their remarks to the Honorary Secretary, and the contributions thus furnished are herewith reproduced.

Dr. E. W. Goodall: I do not think that Dr. Nash need be apprehensive that the ideas put forward in his paper are likely to be regarded as quixotic. In my opinion, he has made out a strong case in favour of the view that the consumption of shellfish has a decided influence upon the prevalence of typhoid fever, in those districts, at any rate, to which he has paid attention. There are one or two points, however, upon which I will ask Dr. Nash to give us further information. Firstly, with respect to the prevalence of typhoid fever at Southend. The notification curve is short and abrupt, and, I understand, corresponds with

the Southend holiday season. Dr. Nash has told us that the sale of shellfish is almost entirely confined to this season. When the season is over, the shops for the sale of shellfish are closed, and are not opened again till the following season. It would therefore appear that these shops are opened chiefly for the benefit of the visitors, and that the visitors are larger consumers of shellfish than are the permanent inhabitants. Does the Southend curve represent the incidence of typhoid fever upon the visitors, or the permanent inhabitants, or both? And if upon the permanent inhabitants, why should they limit their consumption of shellfish to the holiday season? I believe I am right when I say that the bulk of the visitors to Southend come chiefly from the eastern districts of London, and are drawn from classes who can afford to spend only a short holiday at the seaside. If any visitor, therefore, contracted typhoid fever from eating shellfish at Southend, he would almost certainly show no signs of the disease till after his return home. It is therefore important to know to what class of persons the Southend curve refers. I will also ask Dr. Nash whether he hears anything from the Medical Officers of Health of London, or other places, concerning cases of typhoid fever which are reasonably suspected of having originated in Southend.

The second point upon which I ask for further information is with respect to the varying prevalence of typhoid fever in Southend, and the other places for which Dr. Nash has given us figures or curves. It is well known that the prevalence of typhoid fever varies much from year to year. Now if the prevalence depends in any large degree upon the consumption of shellfish, then the sale and consumption of the latter should vary to a like degree. And I ask: Does it? Has Dr. Nash any information upon the sale and consumption of shellfish in different years? Perhaps shellfish are more difficult to procure in some years than in others. Possibly there are local causes which influence the annual consumption.

I think the age-incidence of typhoid fever depends upon other factors than the consumption of shellfish. If the virus of this disease be distributed equally to all the members of a community, where the members are of different ages, the peculiar age-incidence is still well marked. Apparently, there is some personal factor underlying age-incidence.

Lastly, it would be very useful to have some information as to the prevalence of typhoid fever in inland towns and

districts, with reference to the question of its origin in contaminated shellfish. This is a point upon which Dr. Nash has hardly touched in his interesting and suggestive paper.

Lieutenant-Colonel A. M. DAVIES thought that flies were a considerable means of conveying infection in enteric fever, but was not aware that *B. typhosus* had ever been actually demonstrated in or upon the fly; as had been proved in the case of oysters and other shellfish. He had himself endeavoured to isolate the *bacillus* from flies caught over filth trenches in India (where enteric excreta had been deposited), but had only obtained *Proteus* organisms; perhaps *B. typhosus* had been overgrown by the *Proteus*. Subsequent to the outbreak at Shoeburyness referred to by Dr. Nash, there had been a few cases amongst the military, and of these some had appeared to be due to eating infected cockles; in several instances, however, sanitary defects in regard to water supply and drainage arrangements had been present, which made it impossible to positively exclude everything but infected shellfish as the cause of the attacks. No doubt, Dr. Nash had excluded all such conditions in his shellfish cases at Southend. As to seasonal influence, it seemed, as Dr. Nash contended, that the autumnal enteric prevalence could, to a great extent, be explained by shellfish consumption at this time of year, mere temperature having little to do with it; and he believed that in India, where seasonal changes were well marked, it was not the mere changes in temperature and rainfall that were the important factors in increasing or diminishing prevalence, but such means of conveyance of infection as flies and dust, the potency of which, of course, in turn did depend on temperature and rainfall conditions.

Dr. BULSTRODE thought that the members were deeply indebted to Dr. Nash for his interesting and able paper. All were, he thought, in agreement that shellfish eaten raw or imperfectly cooked were liable, under certain conditions, to convey not only enteric fever and, at home, cholera, but also to be the means of setting up very serious attacks of gastro-enteritis. All were, he felt sure, equally in agreement that vigorous steps should be taken to prevent the laying down or collection of edible molluscs in situations exposed to pollution by sewage. The administrative side of the question was, however—as experience had amply demonstrated—beset with considerable difficulties. There was the danger that, by adopting a standard which, in the

eyes of the Legislature would be absolutely impossible of application in practice, such opposition would be aroused that nothing would be done; albeit every epidemiologist would agree that, if possible, every conceivable risk should be obviated.

It was, of course, possible to maintain—and by the adoption of certain bacteriological standards the contention could be supported—that the whole of our littoral, even as far out as the three-mile territorial limit, was at least open to the suspicion of pollution by sewage. If, therefore, a standard of this nature was adopted, and the necessary legislation applied, the result would be the destruction of the whole shellfish industry: not only in so far as this country was concerned, but also with respect to the large trade of imported shellfish. As such a comprehensive step as this would result in at once destroying the livelihood of thousands of persons, it would be difficult in practice to find a Government which would consent to pass the necessary measures. But although there might be administrative difficulties in the way of applying a measure of this nature, there ought not, he thought, to be any difficulty in at once closing beds, layings, ponds or floats, which were obviously exposed to the risks of pollution, and which have been responsible for multiple outbreaks of enteric fever and other illness. It seemed to him that this was the most scientific and utilitarian view to take of the situation, and it was, moreover, a view which there was every prospect of carrying into effect. Having succeeded in closing the more contaminated layings, attention could be directed to places more remotely exposed, with a view to determining how far the evil which obtained could be minimised without the total destruction of the industry. It has, he thought, to be remembered that had this closure of obviously polluted places been carried out several years ago, many lives would have been saved. The difficulty of dealing with shellfish other than oysters was, Dr. Bulstrode thought, very considerable, as a large amount of such shellfish was used as bait by the fishermen. It would, of course, be easy to discriminate on paper between mussels intended for use as baits and those intended for human consumption; but the difficulties of restrictive measures would not be so simple. There was, too, the importation of foreign mussels, both for bait and human food, to be considered. But they must now look to the Royal Commission on Sewage Disposal to solve some of the difficulties he had pointed out. In his view, there was need for more extended research in the direction of the flora of sea-water and molluscs, before

a bacteriological standard could be accepted as the sole basis for the condemnation of either sea-water or shellfish.

Reply by Dr. Nash at the close of the Discussion on his Paper.

I am extremely gratified that my Paper has induced so interesting and instructive a discussion. I agree with Dr. Parsons that some cases of typhoid fever in the autumn are probably induced by eating uncooked vegetables, such as watercress, celery, salads, etc., which have been subjected to specific pollution. In reply to Dr. Goodall, I think the variation in the amount of typhoid fever in different years may partly be explained by the fact that shellfish layings suffer greater specific pollution in some years than in others.

Col. Notter's remarks are very interesting, in view of his exceptional experience in South Africa. I am a distinct believer in the possibility of the evolution of pathogenic properties in commonplace organisms under certain conditions,* and I have ventured in my Paper to-night to state that the increase of organic matter in the London water supply in winter might be a link in the chain of evolutionary conditions in transmitting pathogenic properties to one of the forms of *B. coli*, closely approximating to *B. typhi abdominalis*, which may be present in the intestinal tracts of certain individuals, and who may to this extent be predisposed to typhoid fever. Dr. Butler's remarks practically answer Dr. Parkes; but I may further point out that a careful study of the London diarrhœa curve reveals that in the middle of June the rise in the diarrhœa rate commences from a fairly dead level of about 12 deaths a week, which is the average from December to the beginning of June. In the third week of June the number of deaths has reached 50 in the week. Thereafter the rate of increase rapidly rises, so that the first week in July sees the number of deaths raised to 100, the second week to 150, the third week to 200. For the next fortnight the figure is somewhere about 200 deaths per week. Then it begins to fall, reaching 160 about the end of August, 100 by the middle of September, 50 by the beginning of October, reaching the dead level of about 12

* *Vide* "The Evolution of Pathogenic Organisms," *Medical Press and Circular*, 1901; and "The Evolution of Disease and Disease Germs," *Journal of State Medicine*, 1901.

deaths a week towards the end of November. I suggest that this rise in the diarrhœa curve will be found to closely follow the life-history of the common house-fly: which begins to make its appearance in June, rapidly increases in numbers during July and the beginning of August. Towards the end of August its existence tends to become a mere sexual one. In September the first frost rapidly kills off a large number of flies; and thereafter the insect is found in lessening numbers, until it has practically disappeared by the end of November.*

In reply to Dr. Bulstrode, I maintain that the laying down of any food in sewage-polluted water, no matter how diluted, is an insanitary procedure, seeing that shellfish (especially oysters) are generally eaten uncooked, makes the matter even more indefensible. [Other forms of fish are not eaten raw. They are gutted and cleaned, and then boiled or fried.] Of course the more diluted the sewage effluents the less infection of shellfish; but if the laying of shellfish in the estuaries of rivers is not forbidden, we shall have always to bear the burden of a certain amount of typhoid fever traceable to such shellfish.

I do not presume to have definitely settled the question as to the amount of influence of shellfish on typhoid fever, or of flies on diarrhœa; but I do claim that I have adduced sufficient evidence to justify my belief that they play a not inconsiderable part in the seasonal incidence of these diseases, and that these are matters which require and deserve the closest attention and investigation.

* See "Remarks on the Seasonal Incidence of Diarrhœa and the Part played by Flies," in my contribution to the discussion on Professor Delépine's paper, read before the Epidemiological Society on December 15th, 1902.

THE ETIOLOGY OF LEPROSY.

By JONATHAN HUTCHINSON, F.R.C.S., F.R.S.

(*An Address given May 26th*, 1903.)

Mr. President and Gentlemen,—I have accepted with much pleasure the invitation with which I have been honoured, that I should bring my creed as to the causation of Leprosy under the critical consideration of the Epidemiological Society. That creed is, that Leprosy is a disease the specific cause of which gains access to the system by the stomach, and not by either the skin or the lungs. It admits that any food contaminated by secretions from the skin of a leper may convey the bacillus; but it holds that by far the most frequent mode of initiation is by eating fish which is in a state of commencing decomposition. No precision is attempted as regards the kind of fish or its state, and river fish, sea fish, fresh fish, and salted or dried fish, alike come under suspicion. It is thought possible that the fish may not contain the bacillus itself, but only some toxine which stimulates the tubercle bacillus to activity, and at the same time causes its differentiation. It is fully admitted that these suggestions are only conjectural, and that the evidence upon which the creed rests is circumstantial and not demonstrative. The presence of the bacillus has never been recognised in any form of fish. In explanation of the negative results of such search as has hitherto been made, it is suggested as highly probable that its occurrence (if it be ever present) is very exceptional.

The observations which I have to submit will arrange themselves under two heads: (1) those which discredit the popular theories of contagion and hereditary transmission; and (2) those which give active support to the food hypothesis. I will deal first with some of the facts as regards India.

As a general result throughout India, it would appear that there has been a tendency to diminution of leprosy

during the whole period over which census returns extend. This inference, however, is liable to the fallacy that the original estimate (as it was in Norway and many other places) may have been greatly exaggerated. If we ask as to the causes of the diminution during the last decennium, several plausible suggestions may be made. The Census reporter for Bombay (see p. 166), who reports a reduction from 12,000 to 6,000, attributes it entirely to privation—that is, to famine. Something may have been done by the relaxation on behalf of the Government of the Salt Tax at the large factories, thus permitting of the better curing of fish.

Believers in contagion will allege that the numerous leper asylums which have been instituted throughout India during the last quarter of a century should be credited with some good result, in that they have removed the possible sources of communication of the disease. It may be at once admitted that they may have done something in preventing commensal communication. That they can have had any very large influence is, however, I think very improbable. If we were to suppose that there are 5,000 lepers now under care in asylums in India it would probably be a very liberal estimate. The Commission, twelve years ago, estimated the total leper population at not much short of 100,000; and as the 95,000 still remain at their homes without any sort of supervision, it is obvious that were the disease contagious, very abundant foci still exist.

The statistics as regards increase or diminution in different districts are liable to so much disturbance from famines, formation of leper homes, and other adventitious causes, that it would be an almost fruitless task to investigate them in detail. It may be noted, however, that the increase in Assam has apparently been in connection with the tea plantations and the importation of coolies; whilst in one inland district in Chota Nagpore the disease has increased from four to five in a part where the Roman Catholic population has also increased 15 per cent. (Ranchi). This increase in conversions is quite exceptional, and in adjacent districts there has been a positive decrease in Leprosy.

It is probable, as I have already said, the Leprosy has been decreasing in most parts of India during the last thirty years, that is, from the date of the first Census returns. It is probable also that in many places the decrease has been greater during the last decennium than

in any previous one. The Census figures are, however, open to much suspicion of inaccuracy. The original ones may, and probably did, much exaggerate the numbers, owing to uncertainties of diagnosis, and the inclusion under the head of "white leprosy" of many cases of leucoderma. Of late years the sources of fallacy have undoubtedly diminished, but they are still sufficiently numerous to make it desirable that we should accept their calculations as indications in the direction of truth rather than as trustworthy in detail.

It is universally admitted that throughout India the lower classes and the inferior castes suffer from Leprosy out of all proportion to the higher.

The Brahmins (many of whom eat fish, but some of whom wholly abstain, and all of whom are more or less careful as to their food) suffer very little.

The Jains appear to be almost absolutely exempt. The Commission, in their Report, state that they were informed of one authentic case in a Jain in the Hoshiapur district; but that, apparently, was the only instance of a Jain being infected which they met with. In the tabled list of exceptions at p. 314, 44 Brahmins and 8 Banias are mentioned, but no Jain (see Report, p. 312.)

Districts in India in which there has been Increase of Leprosy.

Census Report for—	Ratio per 10,000 of Population.			
	1872.	1881.	1891.	1901.
Chota Nagpore	2.5	5.3	4.4	5.8
Cachar	4.7	7.8	11.6	—
Assam, with Cachar	6.8	7.0	12.3	—

The explanation of the increase in the Assam territory is easily given. In connection with the tea plantations there has been a large immigration of Coolie labour, and to feed the Coolies a large quantity of salted fish is imported, much of which is of very inferior quality. As to Chota Nagpore, it is not improbable that the early Census was not trustworthy.

Districts in which there has been Decrease of Leprosy.

Census Report for—	Ratio per 10,000 of Population.			
	1872.	1881.	1891.	1901.
Bengal	7.8	9.4	6.2	4.8
Sind	1.4	1.1	0.7	0.47
North-West Provinces	4.2	4.0	3.6	—
South-West Punjab	3.0	2.0	1.0	—

To these the Bombay Presidency may be added, since the last Census has shown a reduction of 50 per cent. This reduction is attributed to famine: in India famine years are always very influential in disturbing the statistics of Leprosy. In the years immediately following the famine there is a great diminution, but some years later this is often more than made up for. The explanation is obvious. During the prevalence of famine many persons are driven to eat food which they would shun at other times.

It may now be convenient to discuss briefly the series of propositions which I have drawn up:—

1st Proposition: That Leprosy never prevails as an epidemic after the manner that all diseases which have been proved to be contagious often do.

In numberless instances the subjects of Leprosy acquired abroad have returned to Europe, or migrated to the United States or elsewhere. In no instance have they caused the disease to spread.

Apparent exceptions to this statement occur in the case of the Sandwich Islands, and perhaps also in New Caledonia.

The facts as regards the Sandwich Islands, and those as to New Caledonia, are, I believe, very similar. There is not the slightest doubt that in both places a very serious, somewhat sudden, and extensive outbreak occurred coincidently with the advent of foreigners. In both instances it has been sought to prove that there was none present before, and thus to strengthen the inference that it was introduced as a matter of contagion, and that it has spread by that agency. If it had been present as an indigenous malady before the foreigners reached the land, then it is obviously open to non-contagionists to ask why did it not spread

earlier? A sudden outbreak implies the introduction of some new influence. It has been proved in the case of the Sandwich Islands that the disease was known to the inhabitants before Europeans came there; but it is certain that it did not prevail extensively. It is exceedingly probable that it was present also in New Caledonia. This island is between the Fijis and New Guinea, in both of which, as well as in many of the closely-adjacent islands, it is fully established that Leprosy has for long been present. It is improbable that New Caledonia was an exception to all its neighbours. That no early mention of the malady was made by missionaries and others may easily be explained by its infrequency, and by the absence of exact medical observation. But, indeed, its presence was asserted by one early authority, and it is only by alleging that he confused elephantiasis with Leprosy that his testimony is set aside. In the Sandwich Islands the outbreak coincided, not only with the advent of Chinese, but with the establishment of a fish-curing factory. Respecting New Caledonia, Le Grand quotes Dr. Rebuffat: "In 1884, seventeen years after the appearance of the leprous Chinamen in the Bondic tribe, one half of the natives, without distinction of age or sex, were affected." This refers to the northern part of the island.

It is highly probable that the Chinese introduced their methods of fish-curing and cookery. An outbreak so sudden and severe is more plausibly attributable to food-poisoning than to contagion. The negative facts as regards contagion are overwhelming.

There is not, I believe, the slightest doubt that not only was the salting of fish introduced as a new art into the Sandwich Islands coincidently with the development of Leprosy, but that the fishing industry received at the same time a great impetus. The use of fish in various forms is now very common there, whereas it was not so formerly.

We may suitably illustrate what I would suggest as the probable explanation of the very remarkable increase of Leprosy in these two places by what we know to have occurred in Cape Colony. There Malay colonies (at Capetown, and some other places) had been introduced by the Dutch with the especial object of fishing and curing fish, and it was near to these establishments that Leprosy was first developed. Instead, however, of attributing its introduction to the Malays, contagionists have been zealous to assert that it came from the Hottentots, although there is no real evidence that the latter had it. It is obvious that if they

had, there could have been no reason why it should not have spread widely before the arrival of Europeans and the introduction of fish eating.

In Cape Colony the disease did not spread rapidly. Nearly half a century occurs after the first mention of cases before the attention of the Government was again called to it. By that time, however, the disease had spread over a considerable area, although still not abundant. Since then it has held its ground and steadily advanced, with a sure but very quiet march to the north and east.

The difference in the rate of progress of the disease in South Africa from that at Honolulu is probably to be be explained by the difference in the local conditions. In the one place, the population is sparingly scattered in small communities, at great distances from each other and from the sources of fish supply. The spread of the disease had to wait, until by degrees roads were made and commercial intercourse was developed. In the Sandwich Islands the very opposite of these conditions prevailed, and a new article of food might come into general use very rapidly. We must remember also that the immigration of Chinese has been continuous and very large, and with them were introduced their habits as regards food and cookery.

If we may make a little allowance for exaggeration in some of the statements made as to the rapidity with which the disease spread both in the Sandwich Islands and New Caledonia, I do not see anything improbable in the supposition of food causation in both instances.

It is noteworthy that in the Sandwich Islands the most ruthless efforts to stamp out the disease by compulsory segregation have utterly failed. Until quite recently the supply of new cases for Molokai has been as abundant as ever, and if it has diminished during the last few years, we are assured that the reduction has been only in ratio with that of the native population.

II.—That the scattered distribution of Leprosy in countries in which it prevails is a fact strongly opposed to the belief in its being contagious.

Leprosy is at present scattered over all India, and over the greater part of South Africa. I take these two countries by way of example. In both of them leprosy continues to be, for the most part, a rare malady. One or two here and there suffer, and nowhere, except at certain fishing villages, does the disease prevail largely. India has only 5 per 10,000; South Africa only 4; and Ceylon less than 2.

Thus there are abundant foci, but nowhere any marked local prevalence. It is inconceivable that a disease which could propagate itself by contagion should be so widely distributed and at the same time so restricted as to numbers.

III.—That its occasional occurrence as a family disease is well explained by the suggestion of commensal communication, and does not support that of skin contagion or inheritance.

There is no doubt that Leprosy often affects more than one member of the same household. On the food theory, this may be explained by family habits and preferences as regards special articles. It is well known that such individual preferences do exist, and it may easily be the fact that several relatives have them in common. If the disease is manifested in early childhood, it may have been contracted by taking food contaminated by the hands of a leper, or by feeding at the breast of a leprous mother. Dr. Hansen has observed that when more than one child in a family suffers, they are usually those born consecutively: a fact which supports these conjectures. If family prevalence were determined by contagion, the whole family would probably suffer.

IV.—That the remarkable persistence of Leprosy in certain districts and amongst certain communities without any material increase, is a fact strongly opposed to the belief in contagion.

The district of Tracadie, in New Brunswick, and certain villages in the Gulf of Bothnia, and in the Russian parts of the Baltic coast, are cases in point. Many other places might be mentioned where the disease persists, but is quite stationary as regards its amount of prevalence. In Iceland, if it were contagious, the whole population might be expected to suffer. In all places, however, where it has been for long prevalent, it continues to affect only a small minority. This implies that it must be due to some cause more or less of accidental incidence.

V.—That the rarity with which husband and wife are both lepers is a fact well-nigh conclusive as regards all forms of contagion.

Vandyke Carter has published some most striking statistics in proof of this, and in it a statement which is confirmed by all observers. It is, I submit, impossible to believe that a disease, in which risk of contagion is supposed to be incurred by the most casual intercourse of social life,

should fail to affect both members of a couple who have lived in conjugal relationship for a dozen years.

VI.—That the general and often great disparity in the prevalence of Leprosy in the two sexes cannot be explained with any degree of plausibility on the supposition that it is a contagious disease.

In a few countries—Norway, perhaps, as an example—the ratio of the sexes is nearly equal, but in the great majority there is a definite and often very great preponderance of males. The countries in which the proportions are most nearly equal are usually those in which poverty is urgent, the supply of cured fish liberal, and no religious scruples intervene. If the supply is limited, men are likely to get the larger share; and it is also an incontestable fact that men are more fond of strong flavours than are women. The latter often express disgust for that which the former will eat with relish. If there be religious prohibition, men are more likely than women to disregard it.

VII.—That there is nothing whatever in the supposed success of segregation measures which supports the belief in contagion.

Norway is the country which we constantly hear cited as showing successful results from segregation. Nothing has resulted from them in the Sandwich Islands, in South Africa, in Minicoy, or in China. In the two latter places they have been in force for many centuries. Their supposed success in Norway is probably quite illusory, and is more probably due to advancing civilisation and greater care in food. Moreover, segregation in Norway is not complete, for one-third of the lepers are still at their own homes. In many other places Leprosy is diminishing quite as fast as in Norway, without any compulsory segregation whatever (Madeira, the Punjab, Bombay Presidency).

VIII.—That the different degree of prevalence in different religious sects, living in the same districts, is inexplicable on the theory that it is a contagious malady.

In India the Jains suffer scarcely at all, the Brahmins and Banias but very little, the Hindoos considerably, the Mahometans moderately, but the Roman Catholics with a severity out of all proportion to the others. Certain Roman Catholic countries—Spain, Portugal, etc.—still suffer considerably. It would be of interest to know whether

there is any district in which Roman Catholic converts have been numerous in indigenous populations where Leprosy has not been the result.

The Census Reporter for Bengal, dealing with the Chota Nagpore district, writes: "The great centre of Roman Catholic missionary enterprise in this province is the district of Rauchi, where its converts exceed 54,000, or about three-fifths of the total number in the province." The increase in the last decennium was 15 per cent. Now, it is precisely in this district that during the last decennium Leprosy has increased from 2 to 3 per cent., whilst in all the adjoining ones it has diminished.

Although it would certainly be unwise in the present state of our knowledge to attach much importance to it, it may not be wholly out of place to ask the attention of the Epidemiological Society to the circumstance that in some districts tuberculosis appears to increase as Leprosy wanes. It would be easy to suggest conjectural explanations, but for the present I will refrain. Both in Iceland and in Norway it would appear that, simultaneously with the decline of Leprosy, tuberculosis has increased. For long it was reputed that there was no phthisis or scrofula in Iceland. It is now recognised that both occur, and not very infrequently. Lupus is said to be still unknown. Although the formerly-asserted absence was no doubt a mistake, yet it may be allowed to prove that the disease was rare. The diagnosis of consumption is easy, and it is impossible that it could have prevailed at all extensively, and yet have been denied to exist.

Dr. Claus Hansen, of Bergen, has stated his opinion that phthisis in the Bergen district has increased 80 per cent. in the last thirty years.

IX.—That Leper Asylums never become foci from which the disease spreads.

I have never heard this disputed; and in many instances, as the result of careful observation, it is strongly asserted.

X.—That in countries where the disease is sparingly prevalent, very usually no precautions are observed, and yet the disease does not increase.

This must have been very remarkably illustrated during the period of decadence of the disease in Britain and in Mid Europe. It is on record that the leper-houses then received many who were not lepers, yet the disease continued to die out. In many parts of India lepers mix

with the general population quite freely, and remain at home with their families. In the out-patients' rooms of all the large hospitals in India lepers may occasionally be seen, yet the disease appears to be on the decline. It is only where the disease is common that, as a rule, the public takes any alarm.

XI.—The absence of Leprosy amongst the Jain Vanis of Bombay Presidency, and the severity with which the Roman Catholics of Salsette and the Hindoo fishermen of the Concan suffer, is a strong if not a conclusive fact in support of the fish hypothesis.

In the following Table, the second column shows the number of lepers which might have been supplied by the population specified, at the average ratio of 5 per 10,000. The third column shows the actual number in the Asylum. A very liberal allowance must be made for the fact that native Christians enter the asylum more willingly than others. It will be seen, however, that the number of Christian inmates is six times that which ought to be found in the entire Christian population (native).

Bombay Presidency. (The Matunga Asylum.)

Religion.	Population.	Calculated Number of Lepers.	Actual Number in the Matunga Asylum.
Hindu	14,197,322	1419	365
Musulman	3,726,594	372	45
Jain	535,950	53	None
Native Christians ...	98,000	10	60

XII.—It is probable that all over the world at the present time Roman Catholics suffer far more than others from Leprosy.

XIII.—The decline of Leprosy in Europe, and its extinction in many large districts, is strongly in favour of its having been formerly caused by fish food.

Few persons in the present day have any conception of the extent to which fish was used as an article of food in

the centuries which preceded the Reformation, or of the completeness of the change which has ensued in our dietetic habits. At present not even the poorest would eat tainted fish, nor, were he willing to do so, would the vigilance of our Sanitary Inspectors allow him to obtain it? Even in Ireland, where Catholicism still largely prevails, salted fish has almost passed into disuse since the introduction of the potato. The following extracts may show what it was in former days in Europe, and what it still is to some extent in Norway where Leprosy still persists:—

From Phil. Robinson's Paper upon "Fishes in Religion," vol. iii, *Fisheries Exhibition Literature*, p. 41, I take the following: "By the Christian religion the consumption of fish is directly encouraged; for, apart from the general precedent afforded by the miracles in Holy Writ, the Church specially enjoins the diet; and this, too, on such a scale that in the time of Queen Elizabeth the annual "fish days" were 145 in number. The chief were the forty days of Lent; the Ember-days at the four seasons, being the Wednesday, Friday, and Saturday after the first Sunday in Lent; the Feast of Pentecost (Whitsuntide), September 14, December 13; the three Rogation-days, being the Monday, Tuesday, and Wednesday before Holy Thursday; and all the Fridays in the year, except Christmas-Day when it falls on a Friday.

"Even after the Reformation the number of fish days continued large; about 1596-7 those observed by the household of Queen Elizabeth being only some thirty-seven days short of half the year." They were made compulsory by law.

The following Table gives the proportion of Leprosy in ratio with the population in certain representative countries:—

—	Lepers, Total.	Ratio per 10,000 Population.
Norway	—	5.0
Iceland	47	7.0
India	100,000	5.4
Ceylon	635	1.8
Minicoy	58	150.0
Cape Colony	600	4.0

From this Table we learn that, excepting under unusual circumstances, the ratio of Leprosy prevalence does not commonly reach 10 per 10,000, and it is for the most part much below it. From the following one we gain information as to what the favouring conditions are.

Excessive Prevalence of Leprosy.	Per 10,000 of Population.
Kaligoan, Balasore, Bengal	500
Minicoy, Maldive Islands	150
Salsette, Christian community ...	50
Fiji	100
Hawaii, Sandwich Islands	400

Kaligoan is a fishing and fish-curing village on the east coast of India, concerning which the facts are supplied in a careful Paper by Mr. Vincent Richards. Minicoy is an island, the inhabitants of which are said to have four fish meals a day. Salsette is a fishing island, and the Christian community is Roman Catholic and observes the fasts. In Fiji the consumption of fish is very large. Respecting the Sandwich Islands I have already said enough. The outbreak of Leprosy followed the development of a fish-curing industry.

BOVINE AND HUMAN TUBERCULOSIS IN INDIA.

A PAPER

CONTRIBUTED BY J. NEILD COOK, M.D., Medical Officer of Health of Calcutta, Honorary Secretary for Bengal.

THE question whether bovine tuberculosis is communicable to man has been a subject of much discussion since Professor Koch announced his disbelief in such transmission, and I believe that the experience of medical men in India will not be without value as evidence in the controversy. For bovine tuberculosis is so rare a disease here that it may practically be left out of count as a factor in the spread of the disease. I have been a medical officer of health in India for more than twelve years, first in Madras and afterwards in Calcutta, and have inspected the carcases and viscera of many thousands of cattle in the slaughter-houses, and also examined the udders of many milch cows without ever discovering a single tubercular case. I have visited the office of the Secretary to the Government of India, Revenue and Agriculture, without being able to procure any report on bovine tuberculosis; and the Inspector-General, Civil Veterinary Department, whom I addressed on the subject, forwarded me the following letter from Dr. A. Lingard, the Imperial Bacteriologist to the Government of India, giving his experience:—

"With reference to your letter, No. 718-412 M., dated 27th December, 1902, enclosing a letter from the Health Officer, Calcutta, I have the honour to inform you that during the last twelve years I have made every endeavour to obtain information with regard to the presence of tuberculosis in cattle in this country. Up to the present time, the only two officers who have sent me answers in the affirmative are:

"(I) Veterinary-Major J. Mills, who has recognised the disease in animals slaughtered for food in the Bombay abattoirs.

"(II) Veterinary-Captain G. H. Evans, who has recognised it in Rangoon.

"I have never came across a single case of tuberculosis in India proper or Baluchistan."

I wrote to Major J. Brodie Mills of the Civil Veterinary Department, who is Principal of the Bombay Veterinary

College, and who has probably had more experience of cattle disease in this country than any other veterinary surgeon. He replied as follows :—

"Tuberculosis is in my opinion a very rare disease in cattle in India, and I speak from an experience of twenty-two years' active work in the country.

"A copy of a paragraph from my Report on the Cattle at the Famine Camps at Ahmedabad is enclosed."

Extract from the Annual Report of the working of the Glanders and Farcy Department in Bombay, dated 16th April, 1900.

CATTLE FAMINE CAMPS.

"There is one point of interest which I consider worthy of record. Out of forty-two post-mortem examinations made by me, I only found signs of tuberculosis in one animal, the seat of the disease being the lungs. This goes far to prove that the cattle of the districts are bred from extremely healthy stock. The absence of tuberculosis, too, can be largely accounted for by the fact that such cattle practically live always in the open : conditions inimical to the propagation of such a scourge."

I also addressed Veterinary-Major Raymond, of the Civil Veterinary Department, Bengal, on the subject; and was favoured with the following statement of admissions of cattle patients into the Bengal Veterinary College Hospital since 1893-94, up to November, 1902-03, and the cases of tuberculosis during each year :—

Year.	No. of Cattle Patients.	Cases of Tuberculosis.
1893 to 1894	53	1
1894 to 1895	312	1
1895 to 1896	234	2
1896 to 1897	203	7
1897 to 1898	360	9
1898 to 1899	503	2
1899 to 1900	633	1
1900 to 1901	699	4
1901 to 1902	526	2
1902 to 1903	632	2
Total	4155	31

I wrote again to Major Raymond, to ask for evidence that the cases described as such were tuberculosis, and received the following in reply :—

"We do not make a speciality of tuberculosis, and, therefore, our notes are not very minute. To the best of my recollection, every case of tubercle actually died in the hospital, except one cow that was removed by the owner. All the cases that died were shown by post-

mortem to have advanced tubercle of the lungs at least, which was all I wanted for diagnosis. In some cases the tubercle bacillus has been searched for and found ; but it has not invariably been searched for. In some cases tuberculin has been used, and gave a reaction ; but it was not used in all cases, because it was not always available. I have not seen a case in the udder, but I must add I have never looked for one."

I must confess that I should have liked more precise evidence on the pathological appearances, the bacteriological examinations, and the tuberculin tests, in these cases; but as Major Raymond has the reputation of being one of the most capable veterinary officers in India, I cannot but accept his statement that a number of cases of undoubted bovine tuberculosis have been admitted to the hospital at Belgachia.

I have since obtained confirmatory evidence of bovine tuberculosis in India from Lieutenant J. Farmer, Civil Veterinary Department, Superintendent of the Government Cattle Farm, Hissar, Punjab, who has given me three years' figures:—

1900	...	...	...	21 cases.
1901	...	...	...	4 „
1902	...	...	...	4 „

I understand that some thousands of cattle are kept at the farm. The diminution in the number of cases would be due to the weeding-out of weakly stock. Lieutenant Farmer informed me that he had seen tubercular mammitis in India. His service has principally been in the Punjab, where severe winters are experienced, so that for several months in the year the cattle do not live so much in the open air as they do further south.

The small amount of attention which tuberculosis has received in India at the hands of the veterinary profession, when compared with such diseases as anthrax, rinderpest, and surra, is, I take it, strong evidence that it is comparatively rare when compared with its prevalence in Europe. I have questioned the Superintendent of the Municipal Slaughter House, Calcutta, who sees about 100,000 carcases of cattle a year, and he informed me that he had never seen "grapes" in the pleura in India. He is the son of an Essex butcher, and was brought up to the trade, so he should have no difficulty in recognising a well-marked case of the disease. In the face of Major Raymond's experience I am inclined to think he may have overlooked cases; but if the disease had been at all prevalent, I do not think it could have escaped attention.

2. The flesh of the ox or cow is not eaten by Hindus, and in some native States it is unlawful to kill cattle for food, though the Mahomedans eat beef when they can get it. Tuberculosis, however, is not more common amongst Mahomedans than amongst Hindus. In the opinion of native doctors with whom I have discussed the point, the reverse is the case.

3. The manner in which dairy products are eaten is practically the same for Hindus and Mahomedans. The first of these is ghee, or clarified butter, which is made by boiling the butter-fats in large iron pans, a process which no tubercle bacillus could survive. Ghee is used in curries and in sweetmeats, and vegetables and cakes of different kinds are fried in it. If milk is drunk as such, it is almost invariably boiled. It is generally taken in the form of curds and whey, which are prepared in a number of different ways, but the milk is almost invariably boiled first. Channa, or curd cheese, is sometimes eaten with sugar, but it is mostly used in the preparation of sweetmeats, when it is mixed with sugar and boiled or fried in ghee.

4. Tuberculosis not being a tropical disease does not appear to have attracted much attention in India. We find even a well-informed medical officer like Dr. Crombie stating at the Berlin Congress that "figures confirm the experience of all medical men in India that, in contrast with that of Europe, diseases of the chest and phthisis amongst them, take quite a subordinate place in the mortality of India."

In Europe most people in at all an advanced state of tuberculosis are seen by qualified medical men, and the disease they are suffering from is for the most part correctly diagnosed, with the result that fairly reliable statistics are obtainable. In India quite the contrary is the case. The majority of the poorer classes are not attended by medical men in their illnesses, and when they die their deaths are registered by people with no knowledge of medicine, or even of the names of familiar diseases. Tuberculosis is commonly spoken of as "fever." Very often malarial fever is followed by tuberculosis; and in such cases the history of malaria, with—it may be—enlargement of the spleen, will mislead a medical man inquiring into the cause of death, the pulmonary symptoms having attracted little or no attention. Again, cases of tuberculosis of the bowels are apt to be mistaken for chronic dysentery, which is a common disease in this country; and fatal cases of respiratory disease, in which dyspnœa is a symptom, are frequently regis-

tered as asthma, which is not in itself a fatal disease. So any statistical records of tuberculosis in India are calculated to be considerably below the actuals. In order to find what a prevalent disease tuberculosis is in India, we must go to the statistics of Presidency towns, where the registration of deaths is somewhat better than it is in the districts, and see the jail returns and those of the troops serving in India, both European and native. For about the last six months every case of death occurring in Calcutta has been inquired into by the sanitary inspectors, nearly all of whom are medical men who have qualified in the local medical college. The returns show about 6½ per cent. of the total mortality as due to tuberculosis. I have been through the recorded histories of the cases, and believe that 10 per cent. would be nearer the mark. Out of 966 consecutive post-mortems done by Captain Leonard Rogers, Indian Medical Service, at the Medical College Hospital, Calcutta, in 13.4 per cent. death was due to tubercle of the lungs, and in 3.6 per cent. to other forms of tubercle. The conditions of life in Calcutta are most favourable to the spread of the disease, for the site of the town is low-lying and alluvial, with a high subsoil water, and the poor people many of them sleep on the ground in overcrowded and ill-ventilated tenement houses or huts. I have seen the earthen floors of the huts covered at night with sleeping forms so that there was nowhere room to put down a foot, though such extreme overcrowding is not general. Besides this, many of the poor natives sleep in the cold weather with no more covering than a thin cotton cloth, though I, a European, sleeping on a bed, require two blankets over me. Add to this insufficiency of nourishing food and long hours of work—for the "sweater" is not unknown in Calcutta—and the disease could not help spreading in such surroundings. The disease, however, is by no means confined to the poorer classes, for practitioners in the town assure me that there is hardly a well-to-do family that has not got its case or cases. Some of the practitioners in the town are of opinion that the disease is much more prevalent than it was a few years ago: which might be expected, as the population of the town has largely increased, and no provision has been made for the additional population. In point of fact, many hutting grounds have been cleared and devoted to other purposes without any provision being made for the people displaced; the principle that a constructive policy must go hand-in-hand with one of demolition not having yet met with general acceptance. Municipal statistics support the view

that the prevalence of the disease is increasing, for, taking periods of five years from 1875, I find that the death-rate from phthisis has nearly doubled, and that from diseases of the lungs has rather more than doubled, in the last twenty-eight years; during which period it has about been reduced by half in countries where sanitary effort has been better applied. I have made recommendations both as regards the housing question and measures for dealing directly with the spread of the infection, and perhaps in time something may be done to check this high and preventible mortality. In Bombay, where aggregation is more intense than in Calcutta, the mortality from tuberculosis appears to be still higher, for the deaths registered under tubercular diseases for 1899, 1900 and 1901, were 6,029, 8,199 and 7,089 respectively. If plague, which was an exceptional cause, be excluded from the total mortality, those figures give a percentage mortality of 12.4 and 17.3 and 14.8 to the total mortality of the town. The following figures, taken from the Jail Reports, are instructive as showing the prevalence of the disease; for unless the sanitary condition of Indian jails is defective, and overcrowding of prisoners the rule rather than the exception, the disease must be contracted in the majority of the cases previous to admission :—

Percentage of Deaths to General Mortality.

	Phthisis Alone.	All Lung Diseases.
North-West Province jails ...	10.25	31.66
Bengal jails	12.82	28.22
Burma jails	19.06	31.16
Andamans Penal Settlement ...	17.2	27.8
All Indian jails	11.64	28.66

From a note on "Jail Tuberculosis" in the *Indian Medical Gazette* for November, 1902, by Dr. Ernest Water, Indian Medical Service, the writer, who is a jail superintendent, expresses his opinion that a certain proportion of the jail tuberculosis is contracted after admission; that it generally affects both the lungs and the bowels; and that it cannot be contracted from food, as the prisoners get neither milk nor beef, and all the food is constantly being examined. Even among healthy young adults in the European and native army, tuberculosis is not an uncommon disease, as shown by the following figures extracted from the report of the Sanitary Commissioner for 1900, which show that in the European army the admissions were 3.7 and the deaths 0.64 per 1,000; in

the native army 4 and 0.87; and in the jail population 9.6 and 5.1 :—

Disease.	European Army in India.								Native Army of India present, 123,463; Enrolled, 146,727.			Jail Population, 121,811.	
	Men, 60,553.				Women, 2,908.		Children. 5,376.						
	Admissions.	Constantly Sick.	Deaths.	Invalids.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Invalids.	Admissions.	Deaths.
Tubercle, not defined ..	..	..	..	..	..	..	..	..	..	..	..	3	3
,, general ..	1	.04	1	..	..	..	2	1	..	..	..	3	2
,, of meninges ..	..	..	..	..	..	..	1	1	3	2	1	1	1
,, of the brain ..		..	..	..	..	..	..	..	..	..	..	1	1
,, of brain and membranes ..	..	.	..	..	..	..	4	3	—	—	—	—	—
,, of the larynx ..	1	.22	—	—	—	—	—	—	—	—	—	—	—
,, of the lungs ..	205	38.71	35	94	11	2	1	..	456	94	70	1029	541
,, of lungs, larynx, and intestines	..	..	..	..	..	..	..	..	..	..	..	1	1
,, of lungs and intestines ..	..	..	..	..	..	.	1	1	..	..	..	38	25
,, of lungs, intestines, and peritoneum ..	..	..	..	.	..	..	..	..	1	1	—	—	—
,, of the lungs and peritoneum ..	..	..	..	..	..	..	..	..	1	1	..	1	1
,, of the lungs and glands ..	..	..	..	..	..	..	..	..	..	..	..	1	1
,, of the intestines	1	.07	..	..	..	..	4	..	..	3	3	58	40
,, of abdomen and peritoneum ..	..	..	..	..	..	..	..	..	..	..	..	1	1
,, of the peritoneum ..	..	..	..	..	..	..	1	..	2	3	..	[illegible]	4
,, of the spleen ..	1	.06	1	—	—	—	—	—	—	—	—	—	—
,, of the lymphatic glands ..	3	.67	..	5	..	..	1	1	31	1	6	31	6
,, of the kidney ..	1	.01	1	—	—	—	—	—	—	—	—	—	—
,, of the spermatic cord ..	..	..	..	..	..	..	..	..	1	—	—	—	—
,, of the testicles	5	.70	..	1	..	..	..	..	1	..	2	—	—
,, of bones ..	1	.06	..	..	..	..			1	—	—	—	—
,, of joints ..	5	1.15	1	1	..	..	2	..	4	1	..	1	1
,, of the spine ..	..	.01	..	1	—	—	—	—	—	—	—	—	—
,, of the skin ..	1	.07	..	..	..	..	..	..	..	..	..	1	—
Total	225	41.77	39	102	11	2	17	7	501	106	82	1177	628

5. Tuberculosis is by no means confined to town populations, for district medical officers have told me of villages where the disease was very prevalent, especially in the northern part of India, where the people sleep in the cold weather in ill-ventilated huts, and lie close together to keep warm; and I have myself seen advanced cases in remote villages in the Himalayas, which have been brought to my tent for treatment.

6. By the kindness of the Principal and Registrar of the Calcutta Medical College, I have obtained records of 649 cases of tuberculosis which have been admitted to the hospital in three years. Of these, only 11 were under 10

years of age: which only proves that tuberculous children were not brought to the hospital for treatment. It may, however, be taken as negative evidence in support of the generally-accepted belief that tuberculosis amongst children is a rare disease in India. The age of most of the patients was between 20 and 40. They appear to be drawn from all the native races, and from all trades and callings, none of the trades with which pulmonary disease is usually associated being noticeably predominant. Six cases were diagnosed as tabes mesenterica, all adults except one, a Eurasian boy of nine. I have tabulated the cases under the following headings:—

Tubercle of the lungs or thorax	509
„ „ lungs and intestines	49
„ „ intestines or peritoneum ...	13
„ „ meninges	7
„ „ brain	1
„ „ larynx	2
„ „ larynx and lungs	6
„ „ glands	20
„ „ spine	13
„ „ bones or joints	26
Tubercular ulcer	3

The Superintendent of the Campbell Hospital, which is a large native infirmary as much as a hospital, kindly gave me a statement of 858 tuberculous in-patients and 118 out-patients, who received treatment in his hospital in three years. With few exceptions, they were diagnosed as tuberculosis of the lungs. Only one of the 858 in-patients was under 10 years of age, 680 being between 20 and 40. The admission of cases of tuberculosis in the Medical College Hospital is discouraged, so the number of cases diagnosed in the wards or shown post-mortem to be tubercle is the more noteworthy.

7. Although the evidence which I have been able to collect is far from complete, and my statistics are wanting in accuracy, I think that they are sufficient to show:

(1) That bovine tuberculosis is found, but is a comparatively rare disease in India; (2) That a large proportion of the population does not eat beef in any form; (3) That the products of the cow are almost invariably sterilised before consumption, both by Hindus and Mahomedans; and (4) that human tuberculosis is a very prevalent disease. So far as the negative evidence I have been able to collect goes, I may add that primary abdominal infection in children is rare in this country; and though the removal of tuberculous cervical glands has been performed at the

Medical College Hospital in a certain number of cases, it is a matter of everyday observation that scars on the neck from glandular abscesses are rare amongst natives—and I might add amongst European and Eurasian children in this country. I am of opinion that this evidence supports the view that there two tuberculoses with which the human subject may be infected: (1) The so-called human tuberculosis, a disease for the most part of adult life, and transmitted principally by the sputum in the form of spray or dust; and (2) the so-called bovine tuberculosis, a disease of infancy and childhood, transmitted from the cow to the human subject by tuberculous milk; and that whilst the human disease is very prevalent in India, the bovine form is rarely met with; and this conclusion appears to be gaining ground in Europe, judging from the articles on the subject which I see in the medical journals.

OBITUARY.

WILLIAM HENRY CORFIELD, M.A., M.D. OXON., F.R.C.P. LOND.

MEMBERS of the Epidemiological Society have heard with sincere regret of the death of Dr. Corfield, their President, during his period of office. Dr. Corfield was not in very good health at the time of his election to the Presidentship, in 1902; but at that time his malady was probably not looked upon as of a serious nature; and he no doubt himself felt that, given fairly good health, he would have more leisure to devote to the interests of the Society than hitherto had been the case: as his retirement from the post of Medical Officer of Health of St. George's, Hanover Square, on the creation of the City of Westminster, had freed him from the trammels of municipal work.

Dr. Corfield, in his youth, underwent an almost ideal preparation for what was to prove the most important subsequent work of his lifetime. His Oxford training in mathematics and natural science, and the opportunities later afforded by his Radcliffe Travelling Fellowship for study on the Continent in the classical schools of learning, no doubt formed the preparation and supplied the stimulus for that devotion to the study of hygiene which was the abiding interest of his manhood. The examination of the Roman aqueducts in the neighbourhood of Lyons, and of prehistoric lake-dwellings in Switzerland, show the kind of study which specially interested Corfield as a student, and which is reflected in much of the work of his later life.

Success came to Corfield early in life. His Oxford career had been a distinguished one, and, shortly after taking his M.B. degree, he was elected to the newly-created Chair of Hygiene and Public Health at University College, London. This appointment he held until his death; and many generations of Public Health students have listened to his lectures, and worked in the hygiene laboratory at University College, which was established under Corfield in 1875.

Much of Corfield's earlier work was in connection with the treatment and utilisation of sewage. In the 'seventies

and 'eighties, and even earlier, the treatment of town sewage had become a very pressing question. An enormous development had taken place in the sewerage of towns, as a result of the labours of Chadwick and the early sanitary reformers. The discharge of crude sewage into the rivers of the country was doing a vast amount of injury, and the best methods of treating and utilising sewage had yet to be ascertained. In 1869 Corfield became a member of the committee appointed by the British Association to inquire into this subject, and for six years worked with characteristic energy as reporter to the committee. His views are fully set forth in his book on *The Treatment and Utilisation of Sewage*, which was first published in 1870, and has subsequently gone through two editions. Corfield was one of those who rendered a great service to sanitarians and the public by pointing out the fallacies underlying the assertions of those who, at the time, were vaunting chemical processes of precipitating sewage and the production of sewage manures from the sludge, as the best and most remunerative methods of utilising sewage. A great many of these processes had been patented, and the public had been invited to assist in the flotation of companies for working these patents, on the assurance that large profits were to be made out of sewage manures.

Corfield's work with the British Association Committee led him to become an ardent advocate of land filtration and of sewage farms. His views on the more modern methods of biological treatment—septic tanks, contact beds, and aerating filter beds—have never, so far as known, been made public. All this recent work has been done since his more active participation in sewage problems ceased; and it seems probable that he was unwilling to express opinions on matters to which his attention had not been actively directed.

Early in his career, Corfield took up the subject of domestic sanitation. In 1879 he delivered the Cantor Lectures before the Society of Arts, on "Dwelling-Houses, their Sanitary Construction and Arrangements," which were subsequently published as a small volume. There was no more able exponent of the true principles of house sanitation than Dr. Corfield, and it is no exaggeration to say that this branch of sanitary science and practice owes a great deal to him. Combined with a strictly logical mind, Corfield had a great capacity for detail and attention to minutiæ, of which he recognised from the earliest time the great necessity in the preparation of efficient schemes of

drainage. With Rogers Field, Corfield shares the honour of being a pioneer in this branch of science, and of being the first to enunciate the true principles underlying house sanitation, and the right methods of their practical application. Corfield's reputation in this respect became great, and he built up an extensive sanitary practice all over the country. Sir William Jenner, at one of the early meetings in connection with the foundation of the Parkes Museum, said that the palaces of the rich were often found by him to be as insanitary as the hovels of the poor. It was largely reserved for Corfield and his fellow-workers to take away this reproach from the rich man's dwelling. His own work as a Medical Officer of Health and that of his colleagues, have done much also to ameliorate the housing of the poor.

Twenty years or so ago, in Corfield's earlier days, the dangers of drain air and sewage gas entering houses were perhaps rather over-emphasised. There was much at that time to warrant the belief that some infective diseases, like enteric fever and diphtheria, and many conditions of ill-health resembling septic poisoning, were the direct results of inhaling sewer or drain air. The more exact knowledge now rendered possible by the science of bacteriology has tended to place a limitation on the injurious effects of defective house sanitation. If Corfield erred at all in attaching too much importance to defective drainage, he erred in good company; he had also constantly before him the astounding results in the improvement of the public health following upon the adoption of better systems of house sanitation—results which could be only attributable to the advances made in the practical application of the laws of hygiene. Even now, we are far from knowing with any certainty the true relations subsisting between insanitary conditions of dwellings and disease outbreaks; and there has been, and probably still exists in some minds, a reactionary disposition to attach too much importance to the bacillus and too little to the conditioning of the soil, to the receptivity of the tissues, and their adaptation as favourable cultural media for bacilli. The predisposing causes of disease were perhaps mistaken for the true causes by the hygienists of the past; but at any rate they were often right in the measures they recommended for counteracting such supposed causes, and on general principles their work is still approved and imitated.

Dr. Corfield's work and his leading position as an English sanitarian were recognised abroad, and he cultivated social relations with many of the most prominent hygienists of

the Continent. His European reputation was of the greatest value when he was acting as Honorary Foreign Secretary to the International Congress of Hygiene, held in London in 1891, and did much to promote the success of that meeting. If the state of his health had permitted, it was the intention of Dr. Corfield, as President of the Epidemiological Society, to have obtained contributions on subjects of special interest to the Society during his term of office from foreign epidemiologists of note; and these contributions would have been very gladly welcomed as tending to widen the basis of the Society's work, and to make it partake more of that International character which is one of its aims and proper functions.

Dr. Corfield acted conjointly with Dr. Netten Radcliffe as Secretary of the Epidemiological Society in the Sessions 1870-1872; and, in 1874, he contributed a paper "On the Alleged Spontaneous Production of the Poison of Enteric Fever," in which he expressed his belief that enteric fever does not arise *de novo*.

In his private life, no less than in his public work, Dr. Corfield was highly esteemed and respected by all. He was ever genial and accessible, and his many interests outside his professional work rendered him a most agreeable companion, free from any touch of that narrowness of view or of judgment, which is sometimes the result of a successful professional life passed in too exclusive a devotion to the daily rounds of duty.

L. C. P.

The Ordinary Meetings of the Society are held at

11, CHANDOS STREET, CAVENDISH SQUARE,

ON THE THIRD FRIDAY OF EACH MONTH FROM NOVEMBER TO MAY INCLUSIVE, UNLESS OTHERWISE ANNOUNCED.

THE CHAIR IS TAKEN AT HALF-PAST EIGHT O'CLOCK P.M. PRECISELY.

Subscriptions may be paid to the Treasurer. The Annual Subscription of Ordinary Members is One Guinea.

Ordinary Members are entitled to all privileges of membership, and to receive a copy of all the Society's publications issued during the year for which their subscriptions are paid. But Ordinary Members, resident beyond the limits of the Metropolitan Postal District, and having paid one or more annual subscriptions, may, on expressing to the Hon. Treasurer a wish to that effect, be distinguished as Non-Resident Members.

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